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ESOPHAGEAL WEBS

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ESOPHAGEAL webs are either thin membranes which sweep partially across the lumen of the esophagus or narrow encircling bands which cause stenosis of its upper portion.

Webs have always been considered to be a rare disease of the esophagus and have neither excited clinicians nor radiologists, which is reflected by the fact that only two articles, which deal directly with webs as an entity, have appeared during the last eight years in the literature, but it should be remarked that webs have been frequently mentioned in connection with the Plummer-Vinson syndrome.

One is, however, surprised when one reads Macmillan's[5,6] report about the roentgenologic findings in a series of 1,600 patients with complaints of dysphagia. He found esophageal webs in 13 per cent of all cases which had positive findings other than foreign bodies accounting for dysphagia. Also, Waldenström and Kjellberg[15] reported 26 cases in 1938 and an additional 12 cases in the first half of 1939, and concluded that webs would probably be found much more frequently if one would search more carefully for them and would be more familiar with the entity.

In 1947, Thomas,[13] in Cleveland, summarized all the existing knowledge on this subject in his article "Webs and Constricting Bands in the Upper Oesophagus (Sideropenic Dysphagia)." He reported 4 cases. All were female patients who had signs or symptoms of essential hypochromic anemia or Plummer-Vinson syndrome. After evaluation of the literature and his own observations he concluded that the primary cause of all symptoms and changes was a primary iron deficiency anemia and that all other changes, including web formation, were secondary to it.

A short article appeared in *Día Médica, —Buenos Aires*, in September, 1953, written by Malenchini and Resano,[7] under the title "Stenosis of the Esophagus Caused by a Congenital Double Web Membrane." It is a simple case report without literature references and does not contribute anything to our present existing knowledge except that the authors were convinced that the described changes were congenital in origin.

ETIOLOGY

The cause of the webs or bands in the upper esophagus is still not definitely known, but it is generally accepted that

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they occur frequently in connection with the Plummer-Vinson syndrome and this is mentioned in all our textbooks. Many authors believe that the epithelial changes in the mouth and pharynx, which are seen in the Plummer-Vinson syndrome, extend into the lower pharynx and into the upper esophagus, and that, finally, atrophic changes which predispose to web formation and fibrosis intervene. However, it is well known that many cases of Plummer-Vinson syndrome show no webs and also that webs may be present without anemia or Plummer-Vinson syndrome. Vinson himself described 69 cases of this syndrome which were all carefully examined by esophagoscopy and roentgen studies, and which showed no evidence of any abnormality of the esophagus.

Webs and fibromuscular thickening may also result from developmental failure. In these cases the obstruction is usually found about the level of the carina and only more rarely in the postcricoid segment of the esophagus, where nearly all other webs are seen. Congenital webs are probably due to a reduplication of the mucosa. It is interesting to find that important textbooks, like those written by Caffey and Schinz, Baensch, Friedl and Uehlinger,[10] do not list webs as a congenital anomaly. Occasionally, webs are seen in elderly patients who have been asymptomatic nearly all their lives and who also do not show any signs of anemia. None of our described theories fits for these cases and, therefore, other causes must exist. Mosher[8] was of the opinion that, while some of the webs are congenital in origin, many of them may be due to scar tissue formation after abrasion of the mucosa as the result of trauma or infection, but his theory does not explain the cause satisfactorily. If his contention were true, we should see webs much more frequently because abrasions of the esophageal mucosa are quite common, and there is no explanation why webs occur practically only in the postcricoid segment of the esophagus. Therefore, we feel that the etiology still remains somewhat obscure.

CLINICAL SYMPTOMS AND SIGNS

Clinically, the chief complaint is dysphagia, which depends partly on the degree of obstruction and partly on spastic factors. Small simple postcricoid webs may cause no symptoms at all. Some patients note only that they have to chew more thoroughly and have to eat more slowly. With rapid swallowing, food and liquids tend to dam back and may cause overflow into the larynx and trachea, resulting in severe paroxysms of choking and coughing. Regurgitation of food is not uncommon and the patients are frequently referred to the radiologic department under the clinical diagnosis of pharyngoesophageal diverticulum. In cases which are believed congenital in origin, it is quite usual that a number of individuals reach adolescence without seeking medical advice. In these cases the lesion is often detected after food impaction has occurred. All patients, however, state that they have always eaten slowly throughout their lives and that they required much longer time for their meals than other poeple.

ROENTGEN FINDINGS

The roentgenologic examination of the upper esophagus is somewhat difficult due to the rapid passage of barium. A relatively thick paste should be used and rapid exposures after swallowing are essential. Webs are easily overlooked and can hardly be recognized on fluoroscopy unless considerable obstruction or retention of barium between a double web membrane is present. Multiple anteroposterior and lateral roentgenograms should be obtained and it is important to realize that webs are usually only seen in the active phase of deglutition, with the lower pharynx and upper esophagus fully distended. The webs appear as thin and sharply defined membranes, which protrude into the lumen from the anterior wall of the lower pharynx or upper esophagus, usually at the level of the cricopharyngeus muscle and rarely below the level of the sternal notch, with the exception of the congenital bands which are often seen at the level of the carina. Oc-

casionally webs surround the entire lumen or appear as a double web membrane. The smooth indentation of the cricopharyngeus muscle should not be mistaken for a web; it always indents the posterior wall of the upper esophagus and the indentation disappears during the Valsalva maneuver.

TREATMENT

In the majority of cases dilatation of the esophagus by bougies is sufficient to enlarge the lumen and is followed by disappearance of the web. Occasionally it is necessary that the web be incised in order to permit passage of the dilators. If anemia is present iron therapy is indicated.

REPORT OF CASES

Case I. Mr. P. W., a sixty-nine year old male patient, was referred to our department because of tickling in his throat and a minimal dysphagia which was present only during the first and second swallow. The symptoms existed for approximately four weeks and the patient stated that he had never had similar symptoms before. He had never experienced regurgitation or vomiting and he did not give a history of having swallowed lye or any other caustic.

Past history: Bronchial asthma since 1943 with mild pulmonary emphysema, otherwise negative. Family history: Negative. Physical examination: Negative, except for mild laryngitis and pharyngitis confirmed by laryngoscopic examination. Red blood cell count, 4,700,000; Hb 104 per cent; color index, 1.1.

The roentgen examination revealed a thin and sharply defined membrane protruding into the lumen of the esophagus from the anterior wall at the level of the cricopharyngeus muscle (Fig. 1). The roentgen diagnosis was simple postcricoid web.

Treatment: Vocal rest and steam inhalations for upper respiratory infection made patient asymptomatic after two weeks. On roentgen reexamination three months later the esophagus was unchanged.

Comment. This case is presented as a typical example of an asymptomatic simple postcricoid web. It is believed that the web would probably never have caused any symptoms and that the dysphagia was most likely due to the mild pharyngitis and to spastic factors during the acute inflammatory stage. The patient had no signs of anemia or Plummer-Vinson syndrome.

Fig. 1. A simple postcricoid web is clearly visible.

Case II. Mr. K. D., a twenty-one year old male patient, was referred to our department after a clinical diagnosis of pharyngoesophageal diverticulum.

The patient stated that he had always had difficulty in swallowing solid food as long as he could remember. Frequently, he was forced to regurgitate, but he stated that he learned to chew very thoroughly and eat slowly. In this way he could control his symptoms quite satisfactorily, but there were occasional exacerbations with choking and coughing paroxysms. He often found it necessary to wash the food down with water or fluids. There was no history of the patient having swallowed lye or any other caustic. He had been examined on numerous occasions, but all examinations were reported as negative. A roentgen examination in 1951 was reported as negative.

Past history: Whooping cough and measles. Tonsillectomy and adenoidectomy at age of five. Family history: negative and noncontributory. Physical examination: Essentially

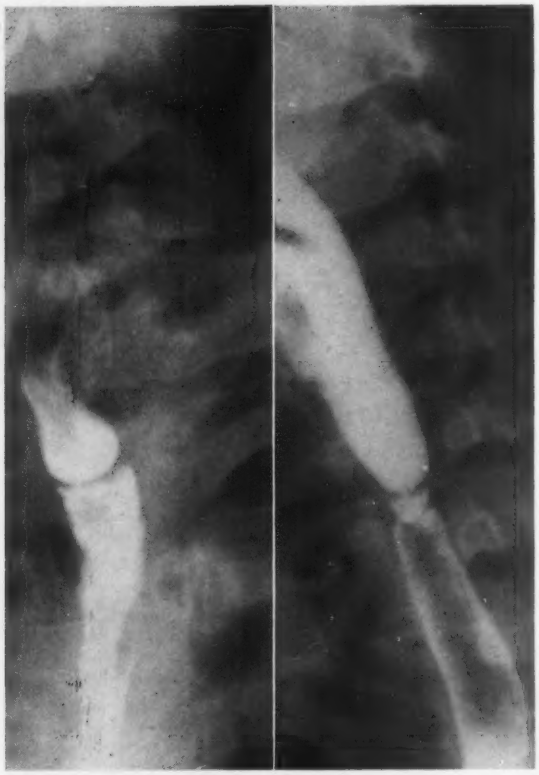

Fig. 2. Encircling and constricting bands in the upper esophagus.

negative. Red blood cell count was 5,500,000; Hb 79 per cent; color index, 0.72.

Roentgen examination revealed an encircling and constricting band in the upper esophagus at the level of C 7. The lumen was narrowed to approximately 3 mm. in diameter. The roentgen diagnosis was stricture of the upper esophagus due to encircling web formation (Fig. 2).

Esophagoscopy (Dr. Ross Robertson) confirmed the roentgen findings and revealed a narrowing of the lumen to approximately 2 mm. in width at the described level. An encircling membrane of approximately 1 cm. in width was seen; it moved freely and there were no signs of inflammation or ulceration.

Treatment: This consisted of dilatation with bougies up to size 32. There were no complications and patient is asymptomatic at the present.

Comment. This case is presented as an example of congenital web formation. The father of the patient is a physician and states that his son has had symptoms since earliest childhood. It appears unlikely that the mild degree of hypochromic anemia which was present had any association with the lesion. The roentgen examination in 1951 was reported as negative, but it is believed that the lesion was not visualized at that time. The upper esophagus was examined fluoroscopically and spot films of the area were taken with inadequate filling at site of the lesion. Roentgenograms failed to demonstrate the web formation but showed only residual barium in the collapsed viscus.

Case III. Mrs. P. A., a twenty-nine year old female patient, gave a history of having difficulty in swallowing as long as she could remember, but she stated that she was told by her aunt that she drank lye which her mother used for scrubbing the floors. She was approximately eight to ten months old at that time. There is, however, no reliable history to indicate that she was ill at that time. She also related that her mother frequently used to take her head between her knees and hit her on the back because she choked so much while eating. Patient had considerable dysphagia and regurgitation of food and had often experienced choking and coughing paroxysms during her meals. She had never consulted a doctor for these symptoms previously because of financial difficulties.

Past history: Usual childhood diseases, including a tonsillectomy and an adenoidectomy, otherwise negative. Family history: Mother died of cancer of the uterus, otherwise noncontributory. Physical examination: Essentially negative. Hb 13 gm.; red blood cell count not known but smear reported as normal.

Roentgen examination showed an encircling web-membrane in the upper esophagus at the level of C 7 (Fig. 3).

Esophagoscopy (Dr. R. H. Gourley) confirmed the roentgen finding. The web was seen somewhat above the sternal notch. It had reduced the lumen of the esophagus to approximately 3 to 4 mm. in width.

Treatment: Dilators were passed without difficulty up to size 34. Then the margin of the web was cut at the right side in order to permit passage of a size 36 bougie. This was easily accomplished but a slight hemorrhage was noted. The patient was returned to the ward in good condition. On the same afternoon she developed fever, and a rise of the pulse rate was noted. A perforation of the esophagus was suspected and the esophagus was explored. The perfora-

tion was readily found, closed, and the mediastinum drained. It was noted that there was no discernible muscle at the site of the stricture. The patient survived and was discharged in satisfactory condition.

Comment. We feel and agree with the clinician that this lesion was in all probability congenital in origin and the story of lye ingestion was most likely a so-called "red-herring." This case emphasized the importance of roentgenographic demonstration of these lesions. Perforation can easily occur at dilatation if the esophagoscopist is not aware of it, particularly if the wall is paper-thin and does not contain any muscle layers.

Case iv. Mr. A. J., a seventy-two year old male patient, was referred to our department because of dysphagia and regurgitation of food. He stated that he had noticed difficulty in swallowing for the last two years. It became gradually worse and, recently, he had experienced frequent regurgitation and also choking while eating. He was particularly troubled when he tried to eat solids but, on a soft diet with thorough chewing and slow swallowing, he was able to control his symptoms fairly well. His appetite was good. No history of weight loss, of having swallowed lye or any other caustic, nor of trauma to the esophagus was presented.

Past history: Bilateral arrested pulmonary tuberculosis since 1930, otherwise negative. Family history: Negative. Physical examination: Negative, except for physical findings in both upper lung fields. Red blood cell count 4,200,000; Hb 83 per cent; color index approximately, 0.99.

Roentgen examination revealed a double web membrane in the upper esophagus at the level of C 7, with slight holdup of barium between the two bands (Fig. 4).

No esophagoscopy had been carried out and

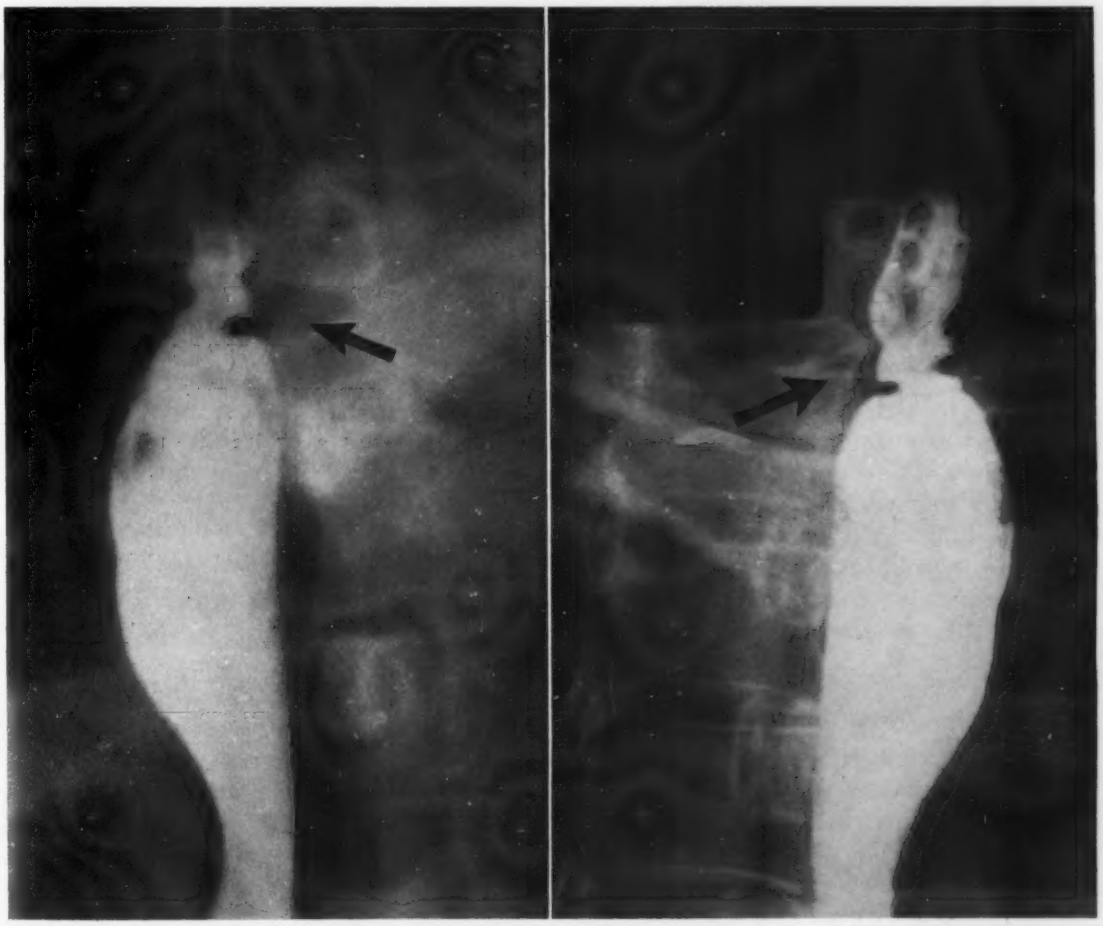

Fig. 3. Narrowing of the upper esophagus due to an encircling web membrane.

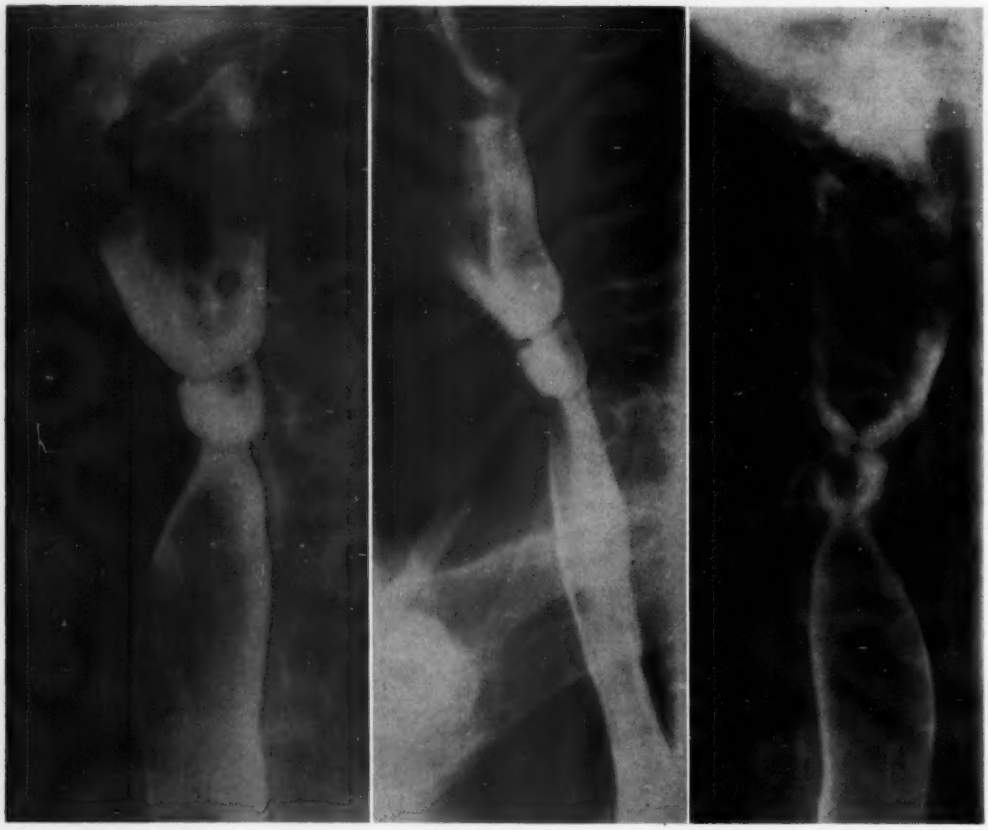

FIG. 4. Double web membranes in the upper esophagus, with hold-up of barium between them.

the patient controlled his symptoms satisfactorily on conservative treatment. The patient was re-examined after six and nine months and no change in the appearance of the esophagus was noted.

Comment. The etiology of this case is obscure. A congenital cause can be considered although the patient had no symptoms for seventy years. He had no signs of Plummer-Vinson syndrome and the mild anemia which was present is normochromic and would therefore not fit into the picture of sideropenic dysphagia.

RELATIONSHIP TO CANCER

Relationship between cancer of the hypopharynx, esophageal webs, and Plummer-Vinson syndrome has been particularly emphasized by the British Authors,[1] by Welin,[16] and others. Whenever a membrane encircles the lumen of the esophagus, it should be regarded as being abnormal for a simple web. Malignant degeneration develops occasionally at the site of the stricture, but is not confined to it and might occur elsewhere in the upper gastrointestinal tract, oral cavity, or hypopharynx. Therefore, careful examination, including the entire esophagus and stomach, should be carried out.

DISCUSSION

We feel that it is not possible to draw any conclusion from a series of 4 cases, but there are a few points of interest.

Webs have mostly been described in connection with the Plummer-Vinson syndrome and iron deficiency anemia. Plummer-Vinson syndrome in males is considered to be a rarity. Three out of 4 patients in our series are male patients. None of them showed signs of Plummer-Vinson syndrome. One had a slight hypochromic

anemia but, it is very doubtful that it was related to the web formation (Case II—most likely congenital). We cannot entirely agree with some authors that webs are secondary to iron deficiency anemia alone. We believe with other authors that webs also occur congenitally. There might be other causes which are not yet known.

The clinician should be careful in making a diagnosis of globus hystericus. The radiologist should not describe an esophagus as negative (if there is a history of dysphagia) before he has obtained satisfactory roentgenograms during the active phase of deglutition with the lower pharynx and esophagus fully distended. Fluoroscopy alone is not sufficient for a thorough examination as barium can pass an esophageal web so quickly as to be indiscernible to the fluoroscopist.

SUMMARY

Four cases of esophageal webs are reported. The etiology is discussed and it is believed that webs occur congenitally, in connection with the Plummer-Vinson syndrome, and in connection with other not definitely known causes. The relationship of encircling membranes to carcinoma of the mouth, pharynx, and upper gastrointestinal tract is mentioned. It is believed that webs are more frequently found when one searches more carefully for them. Whenever a patient complains of dysphagia, roentgenograms of the upper esophagus, during the active phase of deglutition, should be obtained. If the hypopharnyx and upper esophagus are not fully distended during the exposure, webs might be very easily overlooked.

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SPONTANEOUS RUPTURE OF ESOPHAGUS
WITH EMPHASIS ON THE ROENTGENOLOGIC DIAGNOSIS

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MORE than two centuries have passed since Boerhaare[4] described in scholastic detail the first case of ruptured esophagus in the world literature. The first patient with recovery was reported by Frink[6] in 1947. Until that time every case was fatal and in most of them the diagnosis was established at autopsy. Barrett[2] reported the second case in 1947, and in 1948 2 additional cases were described.[14] Our case is being reported because the diagnosis was suggested by chest roentgenograms and confirmed by roentgenologic examination of the esophagus, and the patient was successfully treated by surgery and vigorous antibiotic therapy, with subsequent full recovery.

Rupture of the esophagus is probably the most dramatic of the spontaneous perforations, and early diagnosis in these cases is most important. More than 80 per cent of the undiagnosed patients die in less than 48 hours. The more favorable prognosis of recent years should be attributed to prompt diagnosis, improved surgical techniques, and antibiotic therapy. Curative treatment is not possible without prompt diagnosis and no survival has been reported without surgical intervention.

The so-called "ruptured esophagus" where disease pre-exists should not be considered as spontaneous rupture. In such cases diagnosis is easier because attention is already directed to the pre-existing pathology. This applies also to perforation during esophagoscopy, or stricture dilatation, and to those perforations due to impacted foreign bodies. The term "spontaneous rupture" should include only those cases with no evidence of pre-existing disease. The diagnosis of a perforation in a previously healthy esophagus is missed in most cases because the possibility is not even considered.

The differentiation between true spontaneous rupture of a healthy esophagus and perforation of a diseased esophagus is difficult, and sometimes impossible. For instance, it is known that esophageal ulcers can exist without symptoms or macroscopic evidence of disease, whereas microscopic examination reveals ulceration. On the other hand, artificial perforation of the esophagus in animals by Williams and Boyd[21] demonstrated that if the patient survived several days following the accident the mucosa may show changes in the vicinity of the tear such as infiltration by inflammatory cells due to secondary bacterial infection or due to hydrochloric acid, thus simulating a perforation at the site of previous disease. For these reasons, we feel that Barrett is justified in stating that differentiation is sometimes very difficult and rather of academic interest.

In about 95 per cent of the reported cases, the rupture occurs in the distal third of the esophagus. This is also true in experimental animals. Lerche[10] in his excellent monograph says that in the majority of esophagi the spiral muscle layer extends from the cardia to the upper end of the lower esophageal third, a distance of 8 to 9 cm., which undoubtedly explains the fact that ruptures of the esophagus occur in the longitudinal axis of the organ. A sudden increase of pressure in the esophagus is also an important factor. The exact mechanism *in vivo* has been explained by Mallory and Weiss,[12] who state that with sudden ejection of the gastric contents into the esophagus, the esophagus might fail to relax simul-

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taneously with the diaphragm. Thus gastric contents will be forced with great pressure into a closed tube. Mackenzie[11] states that rupture occurs only when the vomitus cannot be expelled from the esophagus at the same rate as it is received from the stomach. In such circumstances the esophagus is suddenly distended at great pressure and the tube becomes, in effect, a blind pouch, a rupture thus occurring at its weakest point.

The poor coordination in the complicated mechanism of vomiting could very well be due to failure of the sensory center of the vagus, which controls this mechanism. This also could explain the rupture of the esophagus in cases of intracranial lesions. The length of the tear varies from a few millimeters to 8 or 9 cm., but usually is between 3 and 6 cm. More than one tear may occur as in the case here reported. The pressure needed to rupture a healthy esophagus according to Mackenzie's experiments in cadavers is between 5 and 10 pounds per square inch. However, the absence of such factors as muscular tonus and the extrinsic pressure of surrounding organs would cause easier rupture in cadavers than in the living patient. Rupture of the esophagus in children requires almost four times as much pressure as in adults.[13]

If the circumference of the distal esophagus is arbitrarily divided into four quadrants—anterior, posterior, right and left—the rupture is found in the left or anterior quadrants in about 85 per cent of the cases. Rupture in the right quadrant is rare. This has been pointed out by Palmer[15] and Barrett,[1] as well as by others. The explanation accepted is that the right side of the esophagus at its distal end is supported by the adjacent thoracic aorta and the retropericardial fat pad, while on the left side the distal esophagus is adjacent to the parietal pleura. The fact that the intra-abdominal part of the esophagus is almost never ruptured is due to the short stretch of the abdominal esophagus being buttressed by the "liver tunnel" and the fundus of the stomach. This resistance of the abdominal esophagus to rupture has been experimentally shown on both cadavers and animals.

Seventy-five per cent of the patients are males, usually between thirty-five and fifty years of age, the youngest reported patient being five months old and the oldest seventy-nine years. Usually the accident occurs during violent retching or vomiting, although there are a few cases reported in which the rupture happened during routine activity.[15] Acute alcoholism and overfilling of the stomach are considered as predisposing factors.

A diagnostic tetrad emphasized by almost all authors is: (1) Shock, often accompanied by cyanosis; (2) abdominal rigidity, mainly in the epigastrium; (3) mediastinal and subcutaneous emphysema; and (4) hydropneumothorax or hydrothorax.

Cervical emphysema, a very helpful physical finding, occurs in more than 50 per cent of the patients. It is first noted in the supraclavicular and suprasternal regions. The early appearance of cervical emphysema depends upon the direct communication of the ruptured esophagus with the mediastinum. It does not appear until at least two or three hours after the rupture. When there is communication with the pleural cavity, subcutaneous emphysema occurs much later and only after positive pleural cavity pressure has been produced.

The differential diagnosis of a ruptured esophagus should include the following conditions: myocardial infarct, acute pancreatitis, perforated peptic ulcer, spontaneous pneumothorax, pulmonary embolism, mesenteric thrombosis, dissecting aortic aneurysm, diaphragmatic hernia, splenic infarction, acute cholecystitis, rupture of a subphrenic or splenic abscess, ruptured bronchus, volvulus of the stomach and ruptured gallbladder. Of these entities, myocardial infarct, pancreatitis, and perforated peptic ulcer are most commonly confused. In 1955, 3 out of 4 cases reported by Brackney *et al.*[5] were diagnosed as myocardial infarcts, whereas autopsy revealed a ruptured esophagus.

SIGNIFICANCE AND MANIFESTATIONS OF ROENTGENOLOGIC DIAGNOSIS

Differential diagnosis is always a problem even when ruptured esophagus is suspected. Clinical impressions, no matter how well founded, should be verified before surgical intervention is attempted. Many of the reported cases of ruptured esophagus were operated upon for perforated gastric or duodenal ulcer. Often such errors, as well as unoperated fatalities, could be avoided by appropriate examinations. The important roentgenologic manifestations are as follows:

1. Mediastinal emphysema, which is a relatively early sign.
2. Subcutaneous emphysema, generally following the presence of mediastinal air, and first seen deep in the supraclavicular and suprasternal regions.
3. Hydropneumothorax (or pneumothorax), usually on the left, rarely (5 per cent) on the right and not too infrequently (25 per cent) bilateral.
4. Extravasation of contrast material from the esophagus into the periesophageal spaces, the presence of which definitely establishes the diagnosis.

The roentgenograms should be taken in the upright position to exclude the presence of subdiaphragmatic air. Free peritoneal air is not seen in ruptured esophagus, but rather indicates perforation of a hollow viscus other than the esophagus. Preferably, the esophagus should be examined with a water-soluble iodine-containing contrast medium because of the good absorption of the dye, thereby permitting more accurate follow-up examination. In addition, small leakage points may be missed with an oily medium or other high viscosity media. A contrast examination of the esophagus should be done in every suspected case of ruptured esophagus because it is diagnostic, highly accurate, and requires only a few minutes to perform it properly.

REPORT OF A CASE

G.P., a thirty-eight year old colored male, came to the emergency room at 12:30 A.M. on July 6, 1955, complaining of "pain in my belly." The patient had had massive alcoholic intake over the third and fourth of July. On July 5, he felt weak but went to work. On returning home he drank a milkshake. At 7:30 P.M. he had a sudden brief attack of projectile vomiting. In spite of this, he went out and walked several blocks, then returned home and drank a glass of carbonated beverage. He complained of substernal distress for which he took "Tums." At midnight his pain became worse and he came to the University Hospital emergency room.

Past History. Appendectomy, 1940. No history of food allergy or other evidence of sensitivity. No history suggestive of ulcer, pancreatitis, or other abdominal pain similar to present episode.

Physical examination showed the following findings: Blood pressure was 130/70; pulse was 112 per minute; respirations were 40 per minute. The head and neck were negative. The breath sounds were decreased over the posterior right chest, although percussion was equal bilaterally. No rales or rhonchi were present. There was a regular sinus rhythm with no cardiac murmurs. The abdomen showed marked guarding throughout, especially in the midepigastrium, and peristalsis was present. There was no costovertebral angle tenderness. Rectal examination was negative. Extremities were normal. Neurologic examination was not remarkable. The skin was warm and dry, with no lesions. Laboratory data at the time of admission: white blood cell count, 24,000 (86 per cent neutrophils); hemoglobin, 15.2 gm. Urinalysis showed 15 to 20 white blood cells per high power field, with hyalin casts. Serum amylase was 110 Somogyi units. Roentgenograms on admission showed gas in the large and small bowels without distention or fluid levels. There was no free air under the diaphragm. At the time of admission the general impression was (1) duodenal ulcer penetrating into the pancreas or lesser sac, (2) pancreatitis.

A few hours following admission a posteroanterior roentgenogram of the chest and soft tissues of the upper thorax and neck (Fig. 1 and 2) showed air in the mediastinum and subcutaneous emphysema. Roentgen examination of the esophagus was then suggested because a ruptured esophagus was suspected. Under fluoroscopic control the patient swallowed 30 cc. of 70 per cent diodrast, at which time extravasation of the contrast material was noticed at the distal end of the esophagus on the left side a few centimeters above the diaphragm.

Fig. 1. Posteranterior roentgenogram of the chest showing air in the soft tissues of the neck. No evidence of pleural fluid is present, but there is slight clouding of the left lower hemithorax, probably indicating early pleural reaction.

The definite diagnosis of ruptured esophagus was thus established. Fluoroscopically controlled spot roentgenograms were obtained demonstrating the extravasation (Fig. 3). The patient was taken to the operating room approximately twenty hours after the rupture. By then a fever of 103° F. was present, the pulse rate was 144 per minute, and a pleural friction rub was noticed at the level of the seventh and eighth interspaces on the left.

A left posterolateral thoracotomy incision was made and the sixth interspace was entered without dividing the ribs. The left lung was deflated and retracted anteriorly. The inflammatory reaction just anterior to the aorta along the mediastinum could be seen and there was an exudate along the parietal pleura. There was also some exudate on the visceral pleura. A tear measuring approximately 3 cm. was found in the left lateral margin of the esophagus approximately 4 or 5 cm. above the diaphragm. A second rent was located a little more posteriorly but at the same level. No evidence of ulcer, diverticulum, ectopic gastric mucosa or other abnormality was present. Biopsy confirmed this impression. The esophagus was repaired and a large Pezzar catheter was left in place in the left quadrant.

Postoperatively the patient did well. His thoracotomy tube was removed after a sinus tract was established. On July 13, roentgenologic examination of the esophagus showed minimal leakage; therefore, he was continued on his tube feedings. On July 27, a repeat roentgenologic examination of the esophagus showed a small fistulous tract 1 cm. in length, so tube feedings were continued. He was discharged from the hospital on July 28, twenty-two days postoperatively, to be followed by the out-patient department. A roentgenologic examination of the esophagus was performed on August 3, and showed no extravasation. The feeding tube was removed on August 6, 1955. On his visit to the clinic on August 19, 1955, he had gained 4½ pounds, meanwhile remaining on a soft diet. No pain, dysphagia, or vomiting was present. On August 27, he had gained 4 more pounds and was free of symptoms. In November, 1955, he was doing well and had gained 24 pounds since his operation. The most recent roentgenogram, February 27, 1956 (Fig. 4), showed no extravasation or pathologic process. The patient was asymptomatic.

Comment. As with other rare and unusual conditions, the diagnosis of spontaneous rupture of the esophagus will be made only if the possibility is considered and appropri-

Fig. 2. Anteroposterior roentgenogram of the neck demonstrating subcutaneous emphysema.

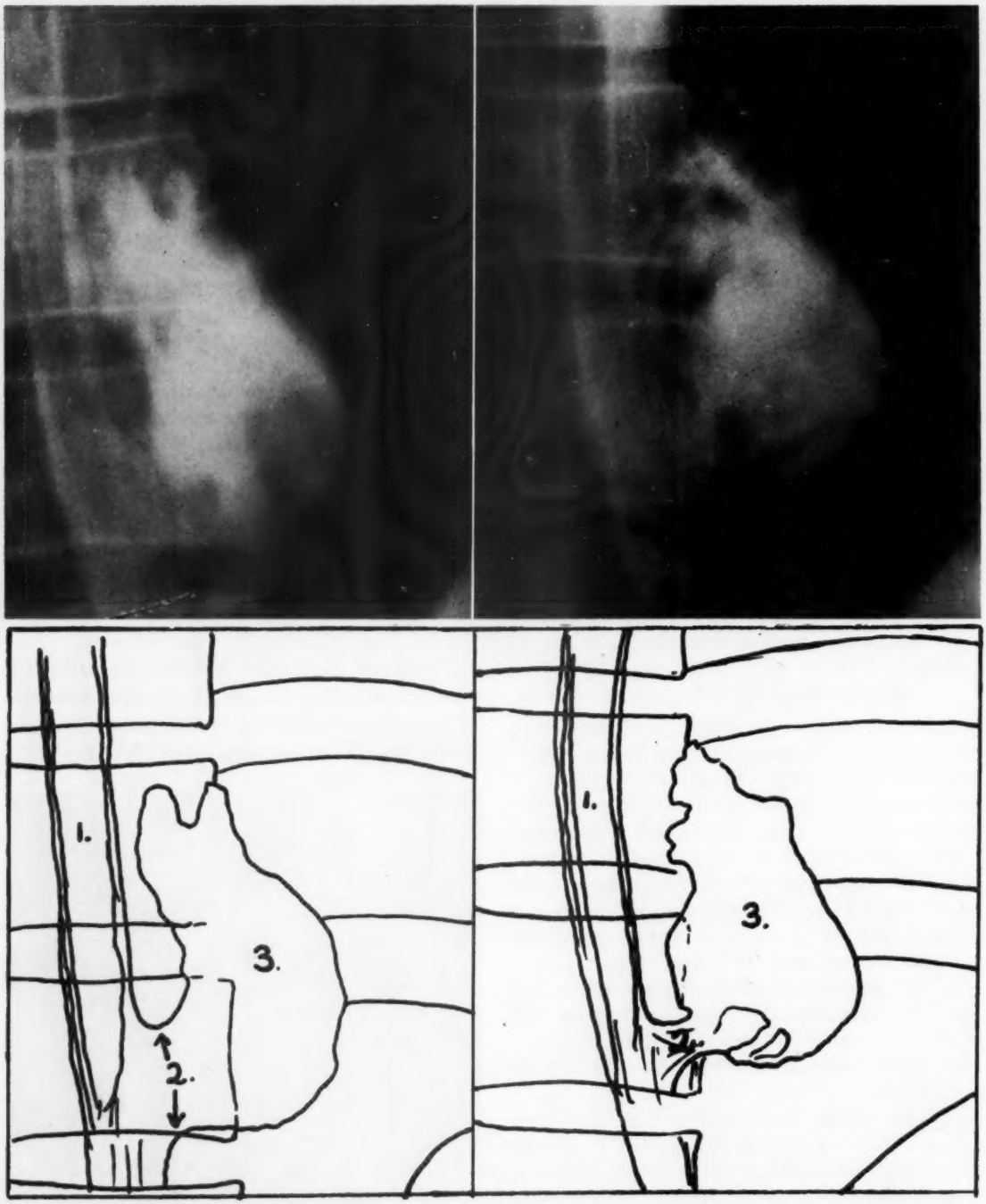

Fig. 3. *Top:* Two spot roentgenograms of the lower esophagus, taken at fluoroscopy, demonstrating extravasation of the contrast material (75 per cent diodrast) into the mediastinum. *Bottom:* Diagram of the spot roentgenograms. 1. Lumen of the lower esophagus outlined by thin streaks of tangentially seen water-soluble contrast material. 2. Contrast material passing from esophageal lumen through the tear. 3. Extravasated contrast material in the mediastinum.

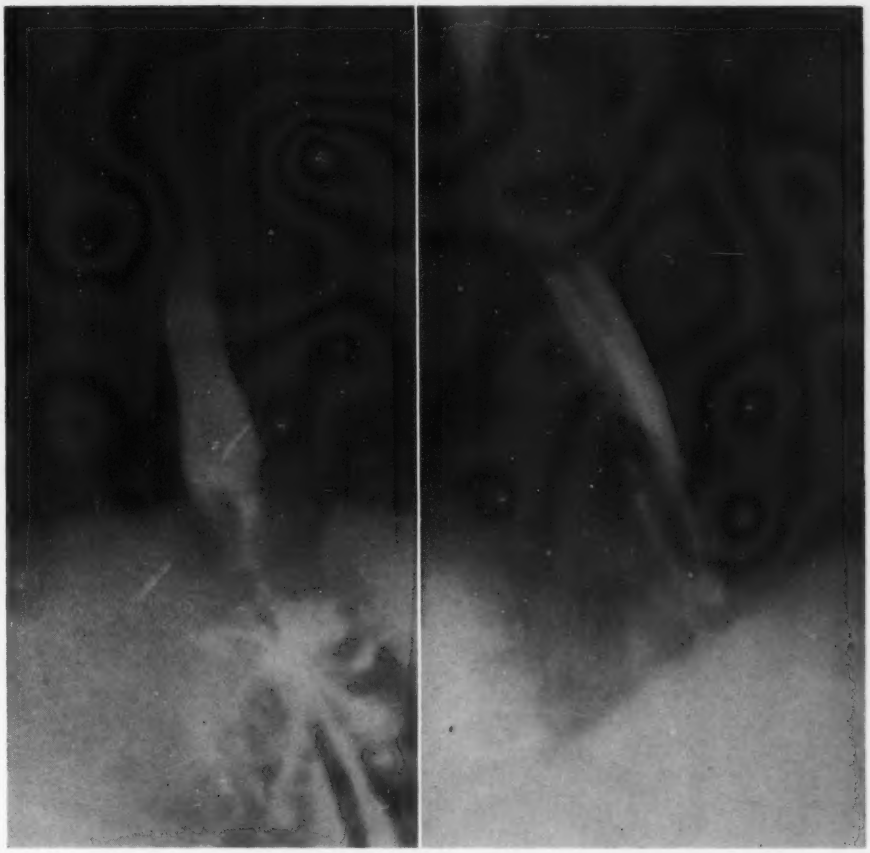

Fig. 4. Approximately eight months after the rupture. No evidence of extravasation.

ate diagnostic procedures carried out. In the reported case direct communication with the mediastinum was present with resultant emphysema. In cases with direct communication with the pleura, emphysema shows up late, if at all, and the etiology of the hydropneumothorax may be easily misinterpreted with disastrous results. It is suggested that every patient suspected of having a ruptured esophagus have a roentgenologic examination of the esophagus, a water-soluble iodine-containing preparation being used. There is no situation in which such an examination might prove harmful.

SUMMARY

A case of spontaneous rupture of the esophagus is reported. The diagnosis was suggested by a chest roentgenogram and verified by means of roentgenologic examination of the esophagus. The patient recovered completely, following repair through a left thoracotomy. The result was confirmed by subsequent fluoroscopic examination with radiopaque contrast material. The history of the disease, the mechanism and the differential diagnosis have been briefly discussed. It is emphasized that the early diagnosis of perforated esophagus can be made roentgenologically and that subsequent vigorous surgical and antibiotic therapy can save the patient's life.

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SIGNIFICANCE OF REGIONAL OR REFLEX ILEUS IN THE ROENTGEN DIAGNOSIS OF CHOLECYSTITIS, PERFORATED ULCER, PANCREATITIS AND APPENDICEAL ABSCESS, AS DETERMINED BY SURVEY EXAMINATION OF THE ACUTE ABDOMEN*

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ROENTGEN examination has proved so informative in the evaluation of acute abdominal disease that it is widely utilized and is considered by many an indispensable part of a diagnostic study. Numerous abnormalities are revealed by plain film examination, including free air in the abdominal cavity due to rupture or perforation of a hollow viscus; calculus disease of the biliary, urinary and pancreatic tracts; abscess in pelvic, appendiceal, subdiaphragmatic and other areas; mechanical, paralytic and vascular occlusive ileus; enlargement and rupture of the liver, spleen and kidney; and finally, tumors.

Minimal requirements for most abnormalities are posteroanterior, anteroposterior and horizontal beam lateral decubitus roentgenograms. The latter are preferred to roentgenograms in the erect position because less exertion and manipulation of the patient are required and ofttimes more information is obtained. Stereoscopic anteroposterior roentgenograms are often superior, and therefore they are included in the examination when indicated. In addition, small cone prone roentgenograms of the gallbladder are obtained when calculus and other disease are suspected. These views are commonly used ones, but conventional lateral and special projections are not infrequently necessary.

In this paper, some roentgen manifestations of cholecystitis, perforated ulcer, pancreatitis, and appendiceal abscess as determined by plain film examination are discussed and illustrated. Frequently these diseases produce reflex or regional ileus as an indirect manifestation of their existence, and therefore this process and its significance will be principally considered. The site of the ileus suggests the area of primary disease. Consequently, significant distention of small bowel in the right upper quadrant warrants a tentative diagnosis of inflammatory disease of the gallbladder or the nearby tissues, and dilatation of a low-lying loop, to the right of the midline merits consideration of appendiceal or pelvic abscess. It is sometimes difficult to be certain of the exact site from this evidence alone because maximal bowel distention is not always found immediately adjacent to the involved viscus, and therefore clinical and other laboratory evidence must be carefully evaluated before an opinion is given. This fact does not lessen the significance of roentgen demonstration of segmental ileus, because frequently it is the most important finding of a nearby inflammatory process.

Reflex ileus secondary to cholecystitis occasionally differs in location from that produced by other nearby disease, such as pancreatitis and a perforated ulcer, in that the bowel beneath the gallbladder shows maximal distention (Fig. 1). Severe peritoneal inflammation may result in partial or complete effacement of bowel markings in the involved segment, and therefore it is sometimes difficult or impossible to determine whether small or large bowel is affected. Occasionally, the dilated loop appears to be fixed, and in some cases it is

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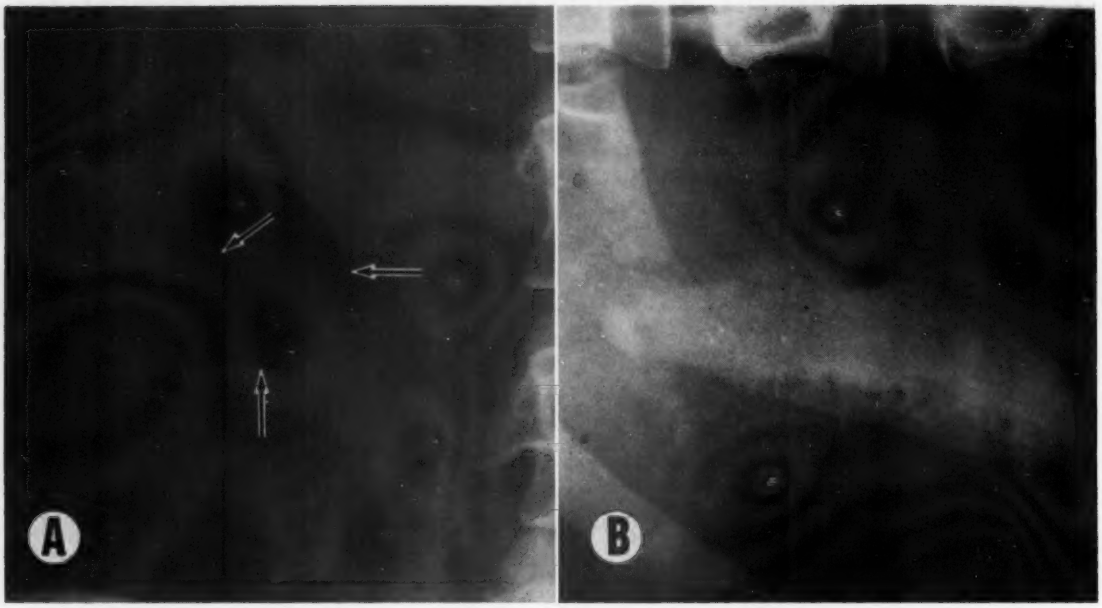

Fig. 1. (*A*) *Reflex ileus secondary to acute cholecystitis* (arrows). Distended loop is probably jejunum because colonic haustral markings are superimposed and adjacent (arrows). Lateral decubitus cholecystogram several days after acute attack shows many nonopaque, small gallstones (*B*).

intimately associated or wrapped around the inferior portion of an enlarged gallbladder. This close relationship contrasts the lower margin effectively and permits a diagnosis of a distended gallbladder with associated ileus (Fig. 2). The jejunum shows involvement more often than the duodenum when the gallbladder is pri-

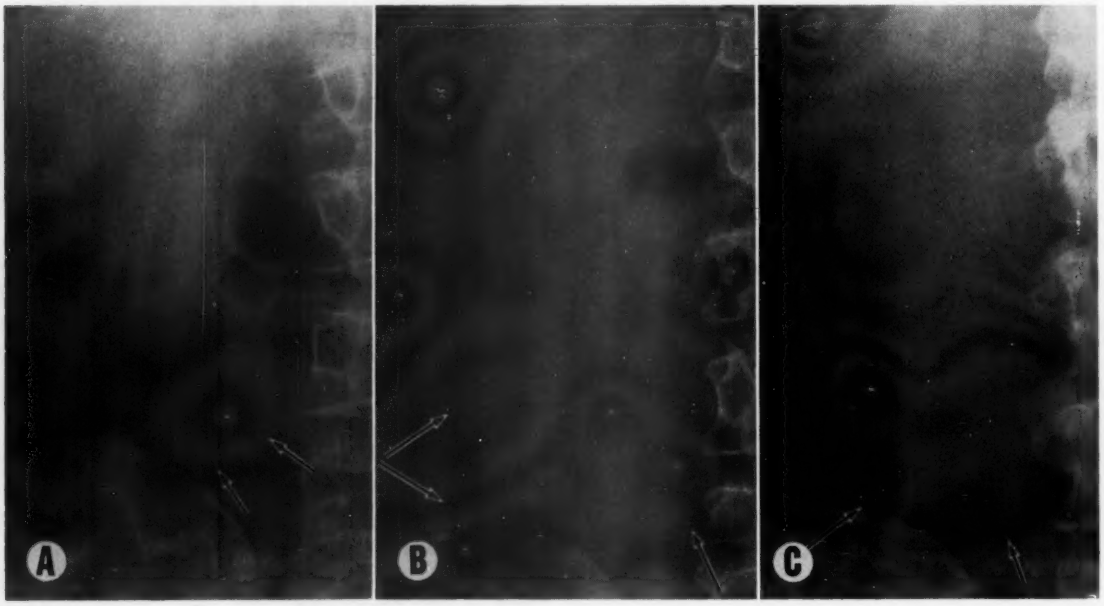

Fig. 2. (*A* and *B*) *Acutely distended gallbladder marginated by neighborhood regional ileus.* (*A*) Medial and inferior walls of enlarged gallbladder outlined by adjacent gas-filled bowel (arrows). (*B*) Inferior edge of enormous gallbladder contrasted by colonic gas (arrows). Large stones impacted in neck of gallbladder obstructed it and caused distention. (*C*) Kidney enlargement simulating distended gallbladder (arrows).

marily diseased. The hepatic flexure and proximal transverse colon are often distended, but this finding is less definitive since gas is commonly seen in the colon. The inferior margin of a distended inflamed gallbladder is frequently outlined by gas in the colon beneath it (Fig. 2*B*). This is due in part to localized ileus, but also because the enlarged viscus indents the colon.

Besides the indirect evidence of acute gallbladder disease furnished by neighborhood ileus, positive information is often obtained by plain film demonstration of an enlarged gallbladder because the viscus and its contents are sufficiently opaque for visualization (Fig. 2, *A* and *B*). An obstructed gallbladder may distend sufficiently to almost fill the right side of the abdomen (Fig. 2*B*). One or more stones may be visible and occasionally one is seen in the neck of the gallbladder. The latter is usually impacted and the cause of the marked distention.

A diagnosis of an acutely inflamed or greatly distended gallbladder is usually justified in a patient with a demonstrable right upper quadrant or right-sided soft tissue mass and clinical findings in keeping with this diagnosis. However, a final opinion should not be given if the kidney shadow and inferior edge of the liver are not clearly defined and distinct from the soft tissue mass. This precaution is necessary because of the unlikely possibility of an enlarged kidney simulating a distended gallbladder in a patient with clinical findings that suggest acute cholecystitis (Fig. 2*C*). When in doubt, immediate intravenous cholecystography, or urography, or both should be done. These procedures take only a few hours at the most, and therefore they do not significantly delay definitive treatment.

Perforation of the stomach or upper small bowel due to an ulcer or other cause may result in ileus. In one patient with severe right upper quadrant pain, ileus of the second portion of the duodenum was found on plain film examination. Subsequent studies led to the diagnosis of perforated duodenal ulcer with localized peritonitis because a small crater was demonstrated in a deformed duodenal bulb (Fig. 3). Other studies including cholecystography were negative. Symptoms were relieved after institution of a strict medical regimen, and subsequent roentgenograms showed no evidence of residual ileus. In another case, segmental or regional ileus involved the duodenum following rupture of a gastric ulcer. The ileus was undoubtedly secondary to localized peritonitis from the ruptured ulcer. The demonstration of reflex ileus is important in the diagnosis of ruptured

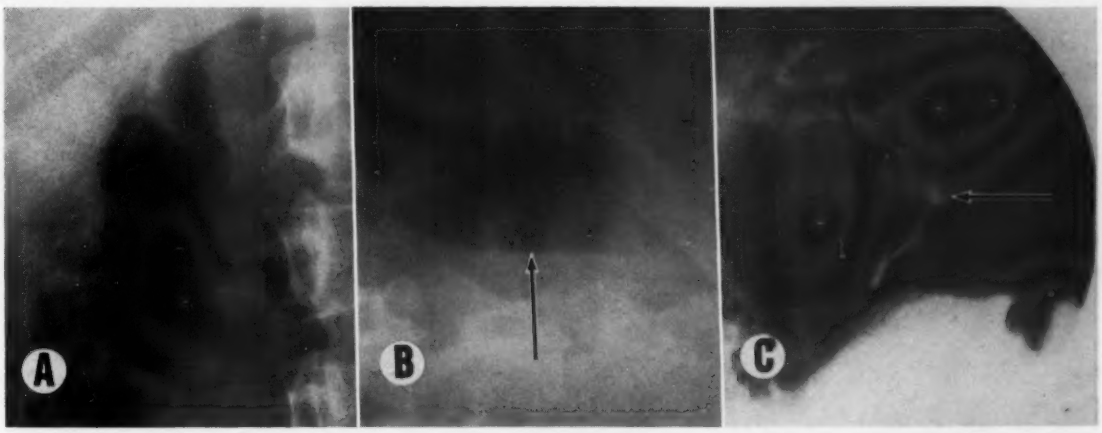

Fig. 3. (*A, B* and *C*) *Duodenal ileus secondary to perforation of ulcer.* (*A* and *B*) Survey roentgenograms reveal excess gas and fluid in first and second portions of duodenum in a patient with acute right upper quadrant pain, and a normal cholecystogram. (*B*) Lateral decubitus view shows fluid level in duodenum. (*C*) Upper gastrointestinal study resulted in demonstration of typical bulb deformity and ulcer crater.

peptic ulcer, especially if free air is not revealed in the peritoneal cavity by horizontal beam upright or decubitus roentgenograms.

Pancreatitis often causes reflex ileus of the regional type, manifested by distention of the duodenal loop and upper jejunum. Proximity of the duodenum to the inflamed pancreas accounts for predominant dilatation of this part of the small bowel (Fig. 4). The descending duodenum frequently contains a disproportionately greater amount of excess gas and fluid, the latter best revealed by a horizontal beam lateral decubitus study (Fig. 4*B*). Greater involvement of the small bowel including the duodenum and jejunum by reflex ileus, secondary to pancreatitis, makes the plain film diagnosis less difficult (Fig. 4). When the peritoneum is diffusely involved, the roentgen findings are less characteristic because reflex or paralytic ileus is more or less generalized. Barry believes that distention of the stomach, duodenum and ascending and transverse portions of the colon occurs frequently in pancreatitis. He found highly suggestive or positive roentgen findings in 12 of 19 proved cases. A study of emergency scout roentgenograms of the abdomen of 125 patients in a series of 307 established cases of pancreatitis led Paxton and Payne[2] to state that segmental ileus was demonstrated in 82 of the 125, or in 86 per cent. In their series, the transverse colon was involved more often than the stomach and small bowel in the later stages of the disease. Their statement that segmental distribution of gas is not pathognomonic but certainly suggests the diagnosis coincides with personal experience. Certainly, a presumptive diagnosis of pancreatitis is justified when plain film examination shows regional neighborhood ileus, and the clinical findings are suggestive or positive. Other adjacent inflammatory disease, including acute cholecystitis, and also mechanical obstruction high in the small bowel must be considered in the differential diagnosis. Calculus disease of the pancreas is not nearly so common as in the gallbladder and urinary tract, but when stomes are present, they are visualized without difficulty. Stones are more commonly demonstrated in chronic pancreatitis.

Appendiceal abscess is sometimes outlined by gas-filled loops of small bowel, or the distended loops may be adjacent to the inflammatory process. For this reason, a perforated appendix should be considered in the differential diagnosis when regional ileus is noted in the right lower quadrant on plain film examination (Fig. 5*A*). Occasionally, there is enough gas in the cecum to show a defect caused by an abscess. When such a defect is detected on initial

Fig. 4. (*A*, *B* and *C*) *Pancreatitis with associated regional ileus.* (*A* and *B*) Duodenum distended in a patient with typical acute pancreatitis and elevated amylase. (*B*) Fluid levels portrayed by lateral decubitus view (arrows). (*C*) Regional ileus involves duodenum and upper jejunum in another patient with acute pancreatitis and a characteristic roentgen appearance.

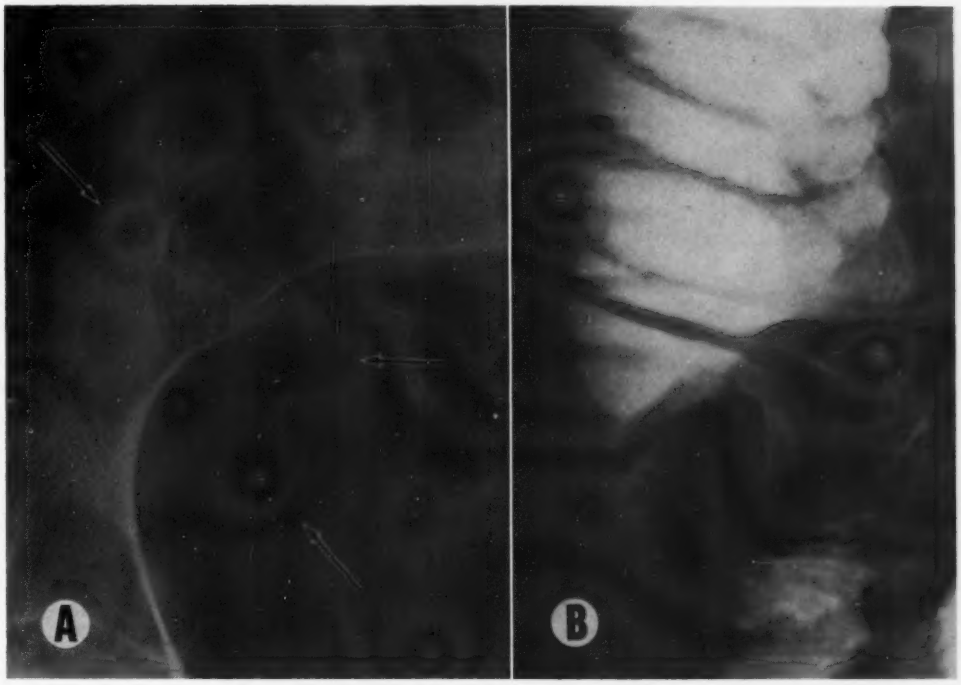

Fig. 5. *Appendiceal abscess demonstrated on survey and barium enema roentgenograms.* (*A*) Reflex ileus from localized peritonitis and displacement effect of abscess on gas-filled bowel are revealed by plain roentgenogram. (*B*) Barium enema roentgenogram shows cecal filling defect due to abscess.

film examination, careful scrutiny of adjacent tissues is imperative for evidence of ileus or increased opacity due to abscess. The abscess is often sufficiently dense for detection without the use of contrast material. Confirmatory evidence can be obtained usually by barium enema examination, and experience to date indicates the procedure is a safe one if done with care (Fig. 5*B*).

The diagnosis of abscess must be considered in a patient with generalized abdominal or right lower quadrant pain when preliminary film examination reveals partial or a high degree of small bowel obstruction. This is true, especially if the bowel is distended as far caudad as the pelvis or to a point near the ileocecal junction, or even beyond this point to an area near the midcecum.

Inflammatory appendiceal changes may manifest themselves anywhere in the abdomen, but roentgen examination, if positive, is usually informative only in demonstrating the site of the appendix unless it perforates and causes an abscess. Information is sometimes obtained about its position by a barium enema or barium meal study, but occasionally gas in the appendix or a calcified appendicolith furnish sufficient contrast to reveal an ectopic appendix. In one patient with pain in the right upper quadrant, plain film examination showed a gas-filled appendix behind the inferior margin of the liver.

CONCLUSIONS

Regional or reflex ileus is frequently demonstrated in the neighborhood of an acute inflammatory process in the abdomen by survey roentgen examination, and therefore it is a valuable indicator of nearby disease.* Location of the ileus suggests the site of involvement, and, in the right upper quadrant, gas-distended bowel often aids in the diagnosis and visualization of an

* In a recent thirty day period, survey film findings of reflex ileus designated the site of acute disease in 5 patients seen consecutively. This localization coupled with clinical and other laboratory findings narrowed the etiologic possibilities so sharply that the correct diagnosis was made without difficulty.

acutely enlarged gallbladder. Fixation of one or two distended loops is additional indirect evidence of neighborhood inflammatory disease and, therefore, a valuable roentgen sign. It is determined by noting absence of appreciable movement on supine, prone, and horizontal beam lateral decubitus roentgenograms. These views are basic requirements and should be supplemented by additional ones when indicated. In this paper, the significance of reflex ileus in the diagnosis of acute cholecystitis, perforated ulcer, pancreatitis, appendiceal abscess and some other plain film manifestations of these diseases have been discussed and illustrated.

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THE ASSOCIATION OF VOLVULUS OF THE CECUM AND ASCENDING COLON WITH OTHER OBSTRUCTIVE COLONIC LESIONS*

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VOLVULUS is a twisting of the intestine causing obstruction and includes torsion of a portion of the bowel about its own long axis or about the axis of its mesentery. Volvulus of the cecum and ascending colon usually presents characteristic roentgen manifestations and in many instances it is possible to establish the diagnosis on the basis of the changes demonstrable on the plain roentgenogram of the abdomen. In 50 per cent of the cases of this lesion observed at the Boston City Hospital, the cecal torsion was found to occur in association with other organic disease of the large bowel distal to the site of the volvulus. Because of the acuteness and severity of the symptoms in most patients with volvulus of the cecum, the concomitant lesion may be masked or its presence remain unsuspected. It is the purpose of this communication to review the clinical and roentgen manifestations on the basis of which the diagnosis can be established and report a series of cases which illustrate the fact that there is a very high incidence of associated disease in the colon distal to the site of the volvulus.

ETIOLOGY

A. Congenital Factors. A prime requisite for the development of volvulus of the cecum and ascending colon is hypermobility of these segments of the bowel. Volvulus usually occurs in association with congenital errors of development and a knowledge of the embryology of the large intestine aids in understanding the pathogenesis of this condition.[2] In the one month embryo, the future intestine is in the form of a simple tubular structure. Shortly thereafter it assumes a U-shape, and a bud-like process which eventually becomes the cecum appears at the distal end of the primitive intestinal tube. At about seven weeks, the developing intestine herniates into the umbilical cord. Approximately three weeks later it re-enters the abdominal cavity and migrates to the right to assume a position in the upper abdomen beneath the inferior margin of the liver. As development continues, the cecum descends into the right lower quadrant, the right mesocolon is brought into apposition with the posterior abdominal wall and the mesentery of the ascending colon fuses with the posterior parietal peritoneum, the process usually being complete by the end of the fifth month. The fusion may fail to ensue, in consequence of which the mesentery remains of sufficient length to permit extensive mobility of the cecum and ascending colon.[34] The cecum normally is capable of a moderate degree of movement and it is only in the presence of marked mobility that volvulus can occur, although many individuals with a mobile cecum never develop this condition. Volvulus of the cecum and ascending colon may occur in association with nonrotation, failure of descent, hyperdescent, and reversal of rotation, a rare congenital anomaly in which the transverse colon lies behind the second portion of the duodenum.

In addition to hypermobility and abnormalities of position of the cecum and ascending colon, there is apparently necessary for the development of volvulus the presence of a point of fixation to serve as a

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fulcrum about which the twisting of the affected segment can take place. This is usually in the ascending colon adjacent to the hepatic flexure, rarely in the proximal portion of the transverse colon or at the inferior margin of the cecum. The torsion occurs in close relation to the point of fixation, which as a rule, is single. Less frequently, it is double with involvement of the ascending colon and terminal ileum. The congenital causes of localized fixation are abnormal pericolic membranes or bands such as the so-called Jackson's membrane[2] which result in marked limitation or absence of mobility of a segment of the ascending colon or the base of the cecum. In the latter instance, the twist involves the ascending colon resulting in an axial type of torsion. This constitutes the "sack type" of volvulus in contradistinction to the more common so-called "loop type" which occurs in the presence of a mobile cecum and a fixed point in the region of the hepatic flexure.[10,12]

B. Acquired Factors. The acquired forms of fixation of the proximal segments of the colon are usually due to postoperative adhesions subsequent to abdominal and pelvic operations or inflammatory changes associated with diverticulitis, pericolitis and pericholecystitis. The torsion and displacement of the cecum and ascending colon may be initiated by one or more precipitating or contributory causes, the most common of which are severe constipation, overeating, ingestion of large amounts of roughage, foreign bodies in the colon, fecaliths, paralytic ileus, marked atonicity, and other states associated with dilatation of the bowel. Pregnancy and pelvic or abdominal tumors which result in displacement of the intestines, surgical manipulation, marked physical activity, direct trauma to the abdomen, diarrhea and violent purgation may serve as etiologic factors. Rarely, a mobile cecum may be an acquired abnormality. In one of our cases, the cecum had been mobilized during an operation for removal of the appendix several years previously and the excessive cecal mobility appeared significant in causing the volvulus which subsequently developed. The presence of a stenosing or obstructing lesion in the distal portion of the colon may initiate volvulus of the cecum and ascending colon, probably because of intestinal stasis and consequent dilatation of the bowel. This occurred in 50 per cent of the cases of our series.

INCIDENCE

The incidence has been variously estimated as from 1–5 per cent of all cases of intestinal obstruction. Volvulus of the cecum and ascending colon is most common in adult life. In our patients, the average age was sixty-eight years, although the lesion has been described in young adults and in persons seventy-five or more years old. There is no apparent sex predilection, some of the previously reported series showing a higher percentage of females, while in others the majority were males. There is a history of previous abdominal surgery in $33\frac{1}{3}$ per cent of our cases of volvulus.

CLINICAL MANIFESTATIONS

While the volvulus of the cecum and ascending colon is relatively uncommon, it is important because of the severity of the symptoms and the high mortality. With greater awareness of the clinical manifestations and the more widespread application of roentgen diagnostic procedures, the number of cases diagnosed correctly and operated upon successfully has shown a striking increase in recent years. Formerly, the lesion was almost always fatal as many patients did not come to operation until serious complications had developed. With the prompt institution of proper remedial measures, the mortality has been reduced markedly and doubtless will show a greater decline in the future. The onset is usually sudden and dramatic. The course of the disease is neither characteristic nor predictable, hence diagnosis is difficult or impossible. The most frequent manifestation is obstipation. There is usually a history of cramps and pain in the abdomen. The pain

may be in the right lower quadrant, the upper abdomen, or generalized. Nausea, vomiting and distention occur in practically all cases. A bulging mass over which there is tympany on percussion is an important diagnostic sign; the bulge may occupy any portion of the abdomen and tends to vary in size and location with the passage of time.

The mesentery becomes constricted in the presence of a marked degree of torsion. There is interference with venous drainage and the arterial supply is compromised, the degree of circulatory impairment being dependent on the vessels affected and the efficacy of the collaterals. The wall of the bowel reflects the changes by the development of cyanosis, congestion, and edema. Complications such as gangrene, infarction, perforation, hemorrhage, and shock often develop rapidly, rendering the prognosis extremely grave or hopeless. The significant clinical signs of infarction and gangrene are tenderness and muscle rigidity in the region of the volvulus, marked elevation of temperature, absence of peristalsis, tachycardia and leuckocytosis. In the presence of gangrene, perforation, or other similar complication, generalized peritonitis may ensue and the symptoms associated with the volvulus tend to be masked.

ROENTGEN MANIFESTATIONS

The roentgen findings in volvulus of the cecum and ascending colon are characteristic, and in most cases the diagnosis can be established with definiteness on the basis of the changes demonstrable on the roentgenogram. The condition is manifested by intestinal obstruction with distention proximal to the site of the torsion. The degree of dilatation is very marked, the affected portion being several times normal size. The cecum, because of the thinness and poor musculature of its walls, undergoes particularly great enlargement. Its outline appears smooth and continuous except for an indentation or interruption at the site of the ileocecal valve, the attachment of the mesentery,[12] or the twist. The distended cecum and ascending colon may be displaced to practically any part of the abdomen and may lie either to the right or left of the midline, in the flank, or in the middle of the abdomen. The fact that the cecum is not in its normal position in the right lower abdomen is an important aid in diagnosis. The haustral markings may be seen if the ascending colon is involved and this establishes the fact that the gas-filled and distended structure visible on the roentgenogram is a segment of the colon, although in some cases the haustrations are so attenuated as to be scarcely recognizable. The ileocecal valve in some instances can be identified as a V-shaped indentation of the border of the cecum, the terminal ileum inserting into the wide portion of the V-shaped defect. The demonstration of this anatomic structure in an abnormal position in association with marked dilatation of the cecum, the ascending colon and the terminal loops of ileum is pathognomonic and establishes the diagnosis with a high degree of certainty. Roentgen studies with the patient in the upright or lateral decubitus positions show one or more fluid levels surmounted by unusually large and radiant gas shadows in the dilated segments of the colon. Serial studies at intervals of hours or days may show increasing dilatation and in some instances variability in the position of the cecum and ascending colon.

Many cases are characterized by axial torsion, the rotation of the affected segment being in the longitudinal axis of the ascending colon. With axial torsion and twisting of 360° or 720°, there is marked dilatation but no apparent displacement of the cecum on the roentgenogram. Volvulus of 180° or 540° is manifested by the presence of the ileocecal junction at the external lateral margin of the cecum rather than in its usual position at the medial aspect. When volvulus is associated with displacement of the cecum in such fashion that the displaced loop assumes a transverse or oblique position, the ileocecal valve lies at the inferior margin of the cecum with a twist of

180° and superiorly with rotation of 360°. In the event that the ileocecal valve is directed anteriorly or posteriorly, identification of this structure is difficult or impossible. The attachment of the mesentery may be demonstrable as an area of increased density and slight narrowing at the medial border of the dilated segment. The actual site of the volvulus may be visualized on the plain roentgenogram of the abdomen as a tapering of the gas-distended colon, although this is unusual. A similar appearance may occur in other obstructive lesions and is not pathognomonic. Absence of dilatation of the transverse, descending and sigmoid colon is a significant diagnostic aid. In the presence of a concomitant stenosing lesion of the colon distal to the site of the volvulus, this manifestation may be absent. Some authors have described cases of volvulus with incomplete intestinal obstruction in which there was considerable gas and fecal material in the bowel distal to the twist.[11]

While the findings on the plain roentgenogram of the abdomen usually prove sufficient to establish the nature of the lesion, the barium enema examination is necessary for diagnosis in some instances. The opaque enema demonstrates which segment of the colon is obstructed. Occasionally, there is visualized the characteristic spiral or corkscrew-like arrangement of the mucosal folds at the site of the torsion. This is not regularly present in every case and the volvulus may be manifested in a smooth, sharply defined manner as with other types of intestinal obstruction. When the opaque enema is administered during the acute stage of the disease, the patient's condition may preclude thorough study and the observer in concentrating on the demonstration of the volvulus is apt to overlook associated colonic lesions. Barium enema examination should not be performed in the presence of gangrene or the roentgenographic demonstration of perforation as manifested by free air under the diaphragm. In $33\frac{1}{3}$ per cent of our cases, gangrene was present, and in $16\frac{2}{3}$ per cent, perforation had occurred. The use of the barium meal is contraindicated in volvulus of the cecum.

In our series of 12 cases of volvulus of the cecum and ascending colon, 6 had an associated lesion of the bowel distal to the site of the volvulus. These comprised 2 instances of diverticulitis, 1 carcinoma of the descending colon, 1 carcinoma of the sigmoid, 1 carcinoma of the rectum and 1 benign stricture of the rectum. The concomitant disease may be silent and its presence totally unsuspected or the manifestations so slight that the patient does not call them to the attention of the attending physician. The symptoms produced by the volvulus are dramatic and severe and other colonic lesions may remain unrecognized for a considerable period of time with the consequent development of serious complications or dangerous sequelae. Because of the high incidence of associated disease of the bowel, complete studies in every patient with volvulus of the cecum and ascending colon are essential to exclude the presence of carcinoma, diverticulitis, or other stenosing process. These examinations are best performed after the patient has recovered from the operation required to relieve the obstruction caused by the volvulus. In addition to the barium enema and double-contrast roentgenographic studies, sigmoidosocpy and digital examination of the rectum should be included. The unusually high incidence of stenosing lesions in the colon distal to the site of the torsion in patients with volvulus of the cecum and ascending colon has not received the attention it merits, although it has been reported previously in the literature.[33]

DIFFERENTIAL DIAGNOSIS

The principal conditions which must be considered in differential diagnosis are paralytic ileus, mesenteric thrombosis, pancreatitis, peritonitis, strangulation of an internal hernia, Hirschsprung's disease, adhesions, neoplasm of the colon and a markedly dilated, obstructed stomach. In paralytic ileus, there is gas throughout the entire gastrointestinal tract, often with multiple fluid levels. The degree of dilatation is

less marked than in volvulus and there is no displacement of the affected loops. Hirschsprung's disease is associated with redundancy and varying degrees of distention. Interspersed throughout the dilated colon are large amounts of fecal material imparting a mottled, irregular character to the densities within the large bowel. Elongation and redundancy of the sigmoid may be associated with a large gas shadow in either the right or left side of the upper abdomen. Barium enema studies make the diagnosis clear. In cases of volvulus with a markedly dilated cecum which is displaced into the left upper abdomen, the condition may easily be confused with a distended stomach such as occurs in pyloric obstruction or acute gastric dilatation. The presence of haustral markings and the globular shape establish the fact that the large, gas-filled structure is comprised of a segment of the colon. However, the haustrations may be obliterated or so attenuated that they cannot be recognized. Studies in the erect or lateral decubitus positions may make identification possible. The passage of a stomach tube or the oral administration of a small amount of barium may be necessary to differentiate the dilated cecum from the stomach. An intra-abdominal abscess subsequent to perforated colonic diverticula or perforation of a gastric ulcer may cause confusion in diagnosis. Reduplication of the colon, ileal volvulus with dilatation of the small bowel, and marked gaseous distention of the bladder may present manifestations similar to those in cecal volvulus. As with other rare and unusual conditions, the diagnosis will not be made unless the manifestations are clearly understood and the possibility of the disease borne in mind. The clinical findings are not characteristic and usually permit only of a diagnosis of intestinal obstruction, roentgen study being essential to determine the site and underlying cause of the obstruction.

REPORT OF CASES

Case I. M. H., a white female sixty-eight years of age, had been having frequent attacks of crampy pain in the right side of the abdomen and vomiting for a period of seven days. The abdomen was distended and tympanitic. Roentgen examination showed a large, gas-filled loop extending horizontally across the mid-abdomen. The left lateral and superior borders of the distended segment of bowel were concave and along its right lateral margin there was an indentation which was considered to represent the ileocecal junction. Multiple dilated loops of small intestine were present in the right upper quadrant of the abdomen and there was absence of the cecal shadow in the right lower abdomen. Operation revealed 180° counterclockwise volvulus of the cecum and ascending colon with marked distention of these portions of the bowel and also of the terminal ileum. Cecostomy and cecopexy were performed. The patient developed a pulmonary infarct and died fifty-one days after the operation.

Case II. S. McG., a white female fifty-one years of age, had been suffering with severe abdominal pain, vomiting and constipation for a period of six days. The abdomen was markedly distended and there was tympany on percussion. An appendectomy had been performed several years previously. On roentgen examination, there was a markedly dilated segment of large bowel occupying the pelvis and abdomen on the right and extending across the midline. There were several loops of small bowel containing moderate amounts of gas in the left upper quadrant of the abdomen. At operation, the cecum was found to be markedly distended, cyanotic, and rotated 180° clockwise. The tip of the cecum was fixed firmly in position by adhesions.

Case III. L. S., a white female seventy-two years of age, had had severe abdominal pain and no bowel evacuations for a period of six days. The abdomen was distended and tympanitic. Peristalsis was absent. A nephrectomy had been performed twenty years previously. Roentgen examination of the abdomen revealed a markedly dilated loop of large intestine occupying the right upper abdomen. The ileocecal junction was visualized at the inferior aspect of the dilated and displaced cecum. The transverse, descending and sigmoid portions of the colon were outlined by gas and appeared moderately dilated (Fig. 1*A*). At operation, there was a volvulus with rotation of 180°, the cecum lying transversely in the mid-abdomen. An adhesive band extending from the cecum to the nephrectomy scar was divided. After the

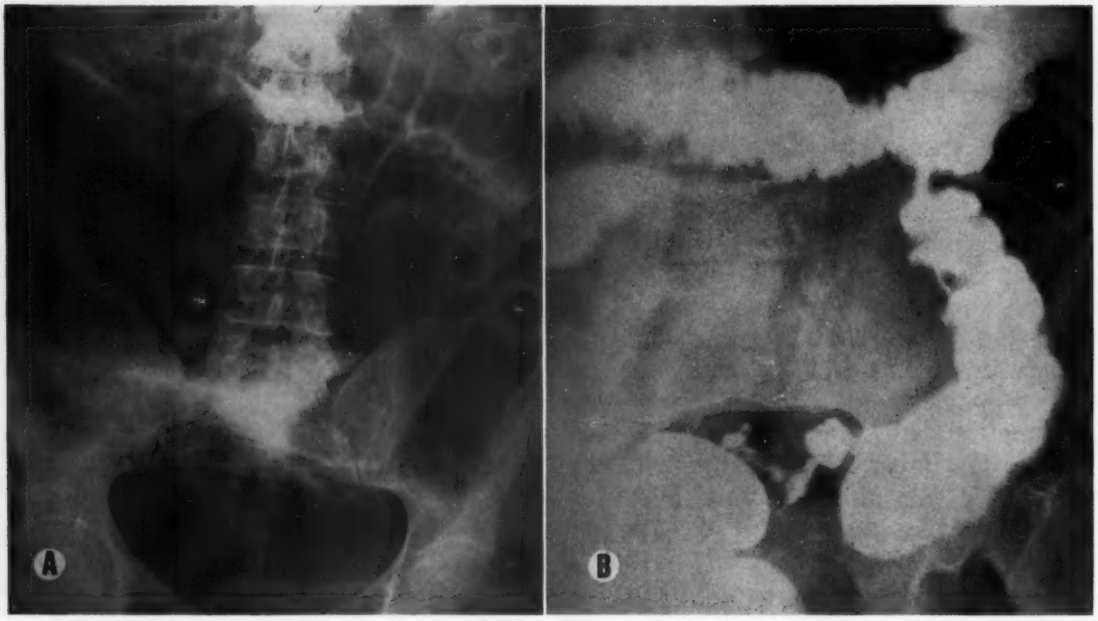

FIG. 1. Case III. (*A*) Plain roentgenogram of the abdomen. The markedly dilated loop of large intestine occupying the right upper abdomen represents the distended and displaced cecum and establishes the diagnosis of volvulus. The transverse, descending, and sigmoid portions of the colon are outlined by gas and are moderately dilated. (*B*) Opaque enema study (after the patient had recovered from the operation for relief of the volvulus) reveals an area of narrowing and irregularity of outline in the region of the junction of the descending colon and sigmoid secondary to diverticulitis.

patient had recovered from the operation, barium enema study revealed an area of narrowing about 4 cm. in length in the region of the junction of the sigmoid and descending colon (Fig. 1*B*). At operation, the stenosis was found to be due to diverticulitis with fibrosis.

CASE IV. L. W., a white female fifty years of age, had had recurring attacks of pain in the mid-abdomen for many years. Four years previously hysterectomy had been performed. Two years later, at laparotomy, the cecum and ascending colon were moderately distended and a diagnosis of chronic diverticulitis with pericolic adhesions was established. The cecum was not fixed. One year ago, a similar attack of abdominal pain occurred. On admission to the hospital, there was abdominal pain, slight distention of the abdomen and diminution of peristalsis. At operation, there was found 360° volvulus involving the cecum, the ascending colon, the proximal portion of the transverse colon and the terminal ileum. The segments involved in the volvulus showed extensive gangrene. Resection and anastomosis were performed. The postoperative course was not remarkable.

CASE V. S. McC., a white female seventy-three years of age, had been suffering for six days with severe pain in the lower abdomen, vomiting and constipation. She appeared acutely ill. There was marked distention of the abdomen, generalized abdominal tenderness and absence of peristalsis. Pulse was 120, respirations 40, temperature 100°F. There was no history of previous abdominal operations. Roentgen examination showed a markedly distended segment of colon occupying the upper and mid-portions of the abdomen with absence of the cecum from its usual position in the right lower abdomen. At operation, there was volvulus of the cecum and ascending colon, the torsion being 180° clockwise. There was a small perforation involving the posterior wall of the cecum. The volvulus was corrected and cecostomy was performed. Opaque enema studies postoperatively revealed a polypoid mass measuring 8 cm. in length in the lower portion of the descending colon with intussusception. The neoplasm was resected and the patient made an uneventful recovery. Histopathologic examination of the mass removed at operation revealed adenocarcinoma, polypoid type.

Case vi. C. N., a white female sixty-three years of age, had a history of intermittent attacks of abdominal cramps for four years. Fifteen years previously, an appendectomy had been performed and thirteen years later, she had undergone a cholecystectomy. Roentgen examination revealed a markedly dilated loop of large bowel occupying the right upper quadrant of the abdomen and extending slightly to the left of the vertebral column. The ileocecal junction appeared to be at the left superolateral aspect of the dilated loop. The terminal ileum was displaced upward and was moderately distended. The right lower quadrant of the abdomen contained no gas shadows (Fig. 2). At operation, there was volvulus, the cecum being displaced medialward and upward into the right upper quadrant of the abdomen. There were multiple adhesions in the region of the cecum and ascending colon. These were divided and cecopexy was performed.

Fig. 2. Case vi. There is a markedly distended loop of large bowel in the right upper abdomen and extending slightly to the left of the midline. There are no gas shadows in the right lower abdomen. The terminal ileum is displaced upward and moderately distended. The roentgen diagnosis of volvulus of the cecum was confirmed at operation.

Case vii. L. McG., a white male seventy-six years of age, for four days prior to admission to the hospital, had been vomiting and had suffered with moderately severe lower abdominal pain. The abdomen was rigid and there was generalized tenderness, diminished peristalsis and hyperresonance. The plain roentgenogram showed a markedly dilated loop of large intestine in the mid-abdomen and the right upper quadrant. Multiple distended loops of small bowel were distributed irregularly throughout the abdomen. The ileocecal junction was slightly to the left of the midline at about the

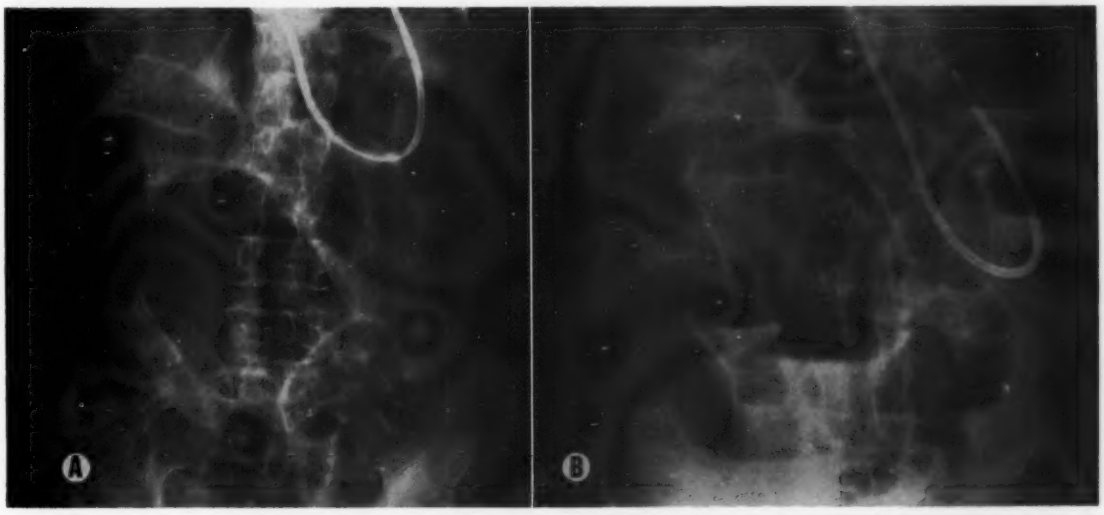

Fig. 3. Case vii. (*A*) Scout roentgenogram of the abdomen. There is a markedly dilated loop of large bowel in the mid-abdomen with multiple distended segments of the small bowel distributed throughout the abdomen and pelvis. (*B*) Same patient. Roentgenogram in the erect position. There are multiple fluid levels. The dilated and displaced cecum lies in the mid-abdomen and the right upper quadrant. Operation revealed a markedly distended and displaced cecum with 360° volvulus, counterclockwise.

level of the iliac crest (Fig. 3*A*). Studies with the patient in the erect position showed multiple fluid levels in the dilated loops in the middle and lower portions of the abdomen (Fig. 3*B*). At operation, the cecum was markedly distended and measured 12 cm. in diameter. There was a volvulus of 360°. The walls of the cecum were markedly thinned. The cecum, ascending colon and proximal portion of the transverse colon were attached to a long membrane in the form of a band. The small bowel had herniated through this band, the hernia involving approximately one-half of the small bowel. The volvulus was corrected and cecopexy and tube cecostomy were performed. The patient recovered without complications or sequelae.

Case VIII. M. S., a white male eighty-three years of age, was admitted to the hospital for treatment of edema and congestive failure due

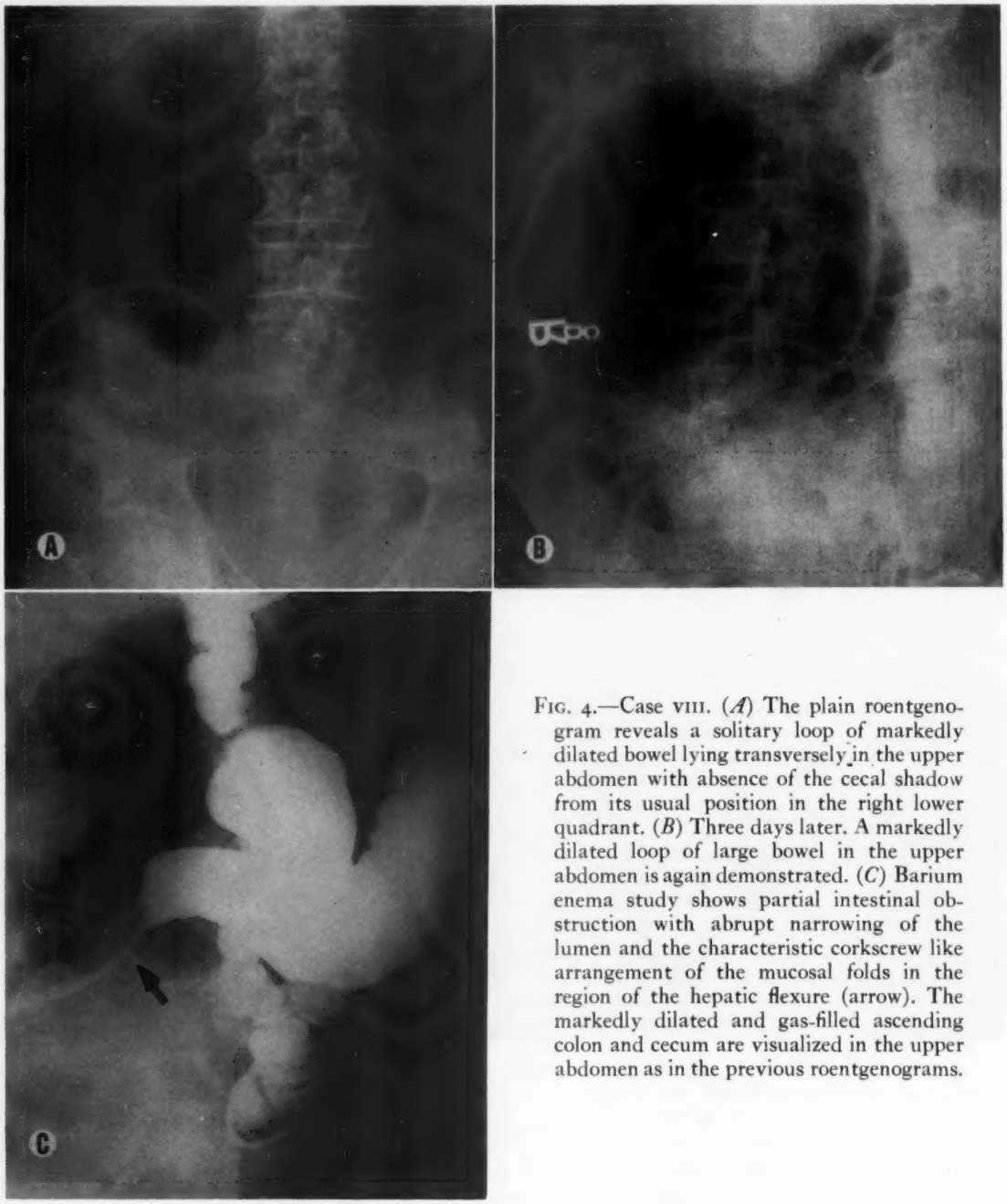

Fig. 4.—Case VIII. (*A*) The plain roentgenogram reveals a solitary loop of markedly dilated bowel lying transversely in the upper abdomen with absence of the cecal shadow from its usual position in the right lower quadrant. (*B*) Three days later. A markedly dilated loop of large bowel in the upper abdomen is again demonstrated. (*C*) Barium enema study shows partial intestinal obstruction with abrupt narrowing of the lumen and the characteristic corkscrew like arrangement of the mucosal folds in the region of the hepatic flexure (arrow). The markedly dilated and gas-filled ascending colon and cecum are visualized in the upper abdomen as in the previous roentgenograms.

to myocardial infarction. While in the hospital he developed severe abdominal distention and obstipation. Twelve years previously, he had been operated on for a hernia and three years later a second operation had been performed for recurrence of the hernia. Roentgenography of the abdomen revealed a solitary, markedly dilated loop of large intestine lying transversely in the upper abdomen (Fig. 4*A* and *B*). The shadow of the cecum was absent from its usual position in the right lower quadrant. An opaque enema examination showed normal filling of the ampulla of the rectum, the sigmoid, the descending colon and the distal half of the transverse colon. In the region of the hepatic flexure, the opaque enema met with almost complete obstruction. The lumen of the colon was narrowed and the characteristic corkscrew-like arrangement of the mucosal folds was clearly evident (Fig. 4*C*). The patient's condition worsened rapidly. Because of the cardiac lesion, operation was considered inadvisable. Death ensued after two days.

Case ix. G. S., a white female seventy-four years of age, for two weeks prior to admission to the hospital, had vomited after each meal and there had been no bowel movements. There was marked abdominal distention, tympany was present over the entire abdomen, and high-pitched peristaltic sounds were heard. Digital examination revealed an area of narrowing in the region of the internal anal sphincter. Opaque enema studies demonstrated volvulus of the cecum and ascending colon in the region adjacent to the hepatic flexure. The opaque enema met with marked delay at the site of the torsion although complete obstruction was not present. The volvulus corrected itself spontaneously and a second barium enema study one week later showed no evidence of narrowing or torsion of the bowel, the entire colon filling without delay at any point. The area of narrowing revealed by digital examination in the region of the rectum was due to a benign stricture and was cured by sphincterotomy.

Case x. I. H., a white female eighty-eight years of age, for four days prior to admission to the hospital, had had obstipation, nausea, and vomiting. The abdomen was distended and there was tenderness on the right. Sigmoidoscopy revealed no abnormalities. The plain roentgenogram showed a markedly dilated loop of large intestine in the right side of the abdomen. The ileocecal junction could be clearly visualized at the lateral margin rather than in its usual position at the medial border of the cecum (Fig. 5*A*). Multiple dilated loops of small bowel were present throughout the abdomen. The transverse and descending portions of the colon were outlined with gas and showed slight

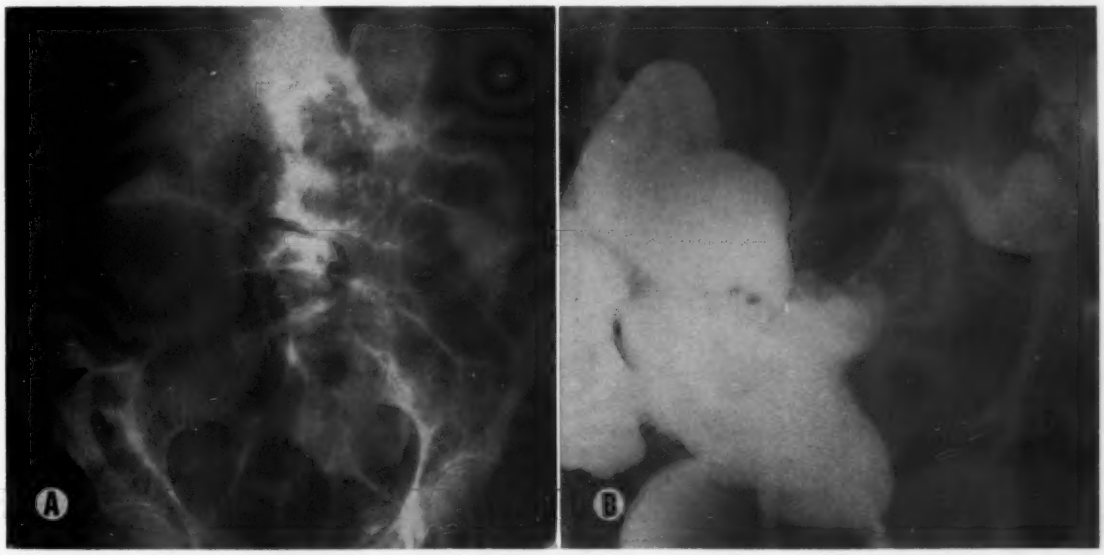

Fig. 5. Case x. (*A*) The plain roentgenogram shows a markedly distended loop of large intestine in the right side of the abdomen and multiple dilated loops of small bowel throughout the abdomen and pelvis. The ileocecal junction lies at the lateral aspect of the cecum (arrow) rather than in its usual position. (*B*) Opaque enema examination demonstrates an extensive area of narrowing and irregularity of outline in the lower portion of the descending colon indicative of carcinoma.

dilatation. Opaque enema examination revealed an extensive area of narrowing and irregularity of outline in the region of the sigmoid and the lower descending colon (Fig. 5*B*) indicative of carcinoma. At operation, the roentgen diagnosis of volvulus was confirmed. The torsion was corrected and cecostomy was performed. The patient expired. Postmortem studies showed carcinoma of the sigmoid, peritonitis, and lobular pneumonitis. (Courtesy of H. A. Osgood, M.D.)

Case xi. G. N., a white male fifty years of age, on admission to the hospital, presented the signs and symptoms of intestinal obstruction with marked distention of the abdomen and obstipation. The plain roentgenogram showed a markedly dilated loop of large intestine in the left upper abdomen. This loop was J-shaped and had the appearance of a dilated stomach (Fig. 6). Multiple markedly distended loops of small intestine were present in the upper and mid portions of the abdomen on the left. The shadow of the cecum was absent from its usual position in the right lower abdomen.

Fig. 6. Case xi. Roentgenogram of the abdomen reveals a J-shaped, markedly distended loop of colon in the upper abdomen, closely simulating the findings in gastric dilatation. Multiple loops of small bowel are present in the upper and mid-portions of the abdomen. The cecum and ascending colon are absent from their usual position in the right side of the abdomen.

Operation revealed a perforation of the cecum and cecostomy was performed, the patient's condition precluding other surgical procedures. The diagnosis of carcinoma of the rectum was established subsequently and a Myles resection was done.

Case xii. A. O'C., a white female sixty-three years of age, while in the hospital for treatment of lobar pneumonia involving the right lower lobe, developed marked abdominal distention, nausea, and vomiting. A plain roentgenogram of the abdomen showed a markedly dilated loop of large bowel extending horizontally across the upper abdomen. The cecum occupied the left upper quadrant and was distended to several times normal size. Several moderately dilated loops of small intestine were present in the upper and mid-portions of the abdomen. The findings were considered characteristic of volvulus of the cecum and ascending colon. The following day, operation was performed. There was 180° volvulus of the cecum and ascending colon, the cecum lying in the left upper quadrant of the abdomen. Incipient gangrene of the ileum was present but the patient's condition precluded resection. Detorsion and cecopexy with tube cecostomy were performed. The patient expired three days later.

In our series of 12 consecutive cases of volvulus of the cecum and ascending colon, 10 of the patients were operated on, of whom 3 expired; gangrene of the bowel developed in 4 instances; and perforation was present in 2 cases. Only 1 of those with gangrene died and no fatalities ensued in the patients with perforation. Of the 2 individuals who were not operated on, 1 expired, while in the second there was spontaneous correction of the volvulus. Of the 6 patients with an associated lesion in the colon distal to the site of the volvulus, 3 had evidences of gangrene of the bowel at the time of the initial operation, perforation had occurred in 2, and there was 1 death.

SUMMARY

Volvulus of the cecum and ascending colon occurs in the presence of hypermobility and abnormalities of position of these portions of the bowel. The congenital and acquired factors associated with the

Table I

Case	Age	Sex	History of Previous Abdominal Operation	Gangrene	Perforation	Operation for Relief of Volvulus	Died	Associated Lesion of Colon
I	68	F	No	No	No	Yes	Yes	No
II	51	F	Yes	No	No	Yes	No	No
III	72	F	Yes	No	No	Yes	No	Yes
IV	50	F	Yes	Yes	No	Yes	No	Yes
V	73	F	No	Yes	Yes	Yes	No	Yes
VI	63	F	Yes	No	No	Yes	No	No
VII	76	M	No	No	No	Yes	No	No
VIII	83	M	No	?	?	No	Yes	?
XI	74	F	No	No	No	No	No	Yes
X	88	F	No	No	No	Yes	Yes	Yes
XI	50	M	No	Yes	Yes	Yes	No	Yes
XII	63	F	No	Yes	No	Yes	Yes	No

hypermobility and abnormalities of position are described. A point of fixation is apparently necessary to serve as a fulcrum about which twisting of the mobile segment can take place. While the site of fixation is usually adjacent to the hepatic flexure, congenital pericolic membranes or acquired adhesions may produce fixed areas in the more proximal portions of the colon. The incidence of volvulus of the cecum and ascending colon is highest in adult life and in more than 50 per cent of the patients in our series there was a history of previous abdominal operations.

The clinical course in patients with volvulus of the cecum and ascending colon is neither characteristic nor predictable. Obstipation is the most significant manifestation and is associated with other evidences of intestinal obstruction. A bulging mass over which there is tympany on percussion is an important diagnostic sign. Complications such as gangrene, infarction, perforation, hemorrhage and shock may develop rapidly.

The roentgen findings in volvulus of the cecum and ascending colon are characteristic and usually the manifestations on the plain roentgenogram of the abdomen suffice to establish the nature of the lesion. If the diagnosis cannot be made with definiteness from the scout film, barium enema studies of the colon reveal the point of obstruction and in most cases demonstrate its cause. The importance of early and accurate diagnosis and prompt surgical intervention is emphasized.

Two related concepts are stressed in our presentation. The first is the presence of an unusually high incidence of lesions in the colon distal to the site of the volvulus. This occurred in 50 per cent of our patients, there being 2 cases of diverticulitis of the colon, 1 of carcinoma of the descending colon, 1 of carcinoma of the sigmoid, 1 of carcinoma of the rectum, and 1 of benign rectal stricture. The second consideration is that special studies including particularly a barium enema examination, double contrast roentgenography, and sigmoidoscopy should be performed in all cases of volvulus of the cecum and ascending colon as soon as the patient's condition permits. In most instances these studies are best done postoperatively after rectification of the volvulus.

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Vol. 78, No. 4

DIFFUSE CALCIFICATION OF THE PANCREAS*

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SINCE 1937, 18 cases of diffuse calcification of the pancreas have been diagnosed in the Department of Radiology of the Philadelphia General Hospital. Two of these cases were previously reported.[11,27] A review of the literature shows 77 cases recorded (Table 1), and the 16 new cases reported herein bring the total to 93.

Mayo[15] in 1936 described 2 types of calcium deposits within the pancreas. The first "true stones" he described as concretions of calcium salts lying free in the ducts, these stones being single or multiple and usually in the larger ducts. The second variety he called "false stones," or the so-called actual calcifications of the parenchyma of the pancreas. Beling[2] in 1942, stated that the pancreas may be the seat of diffuse parenchymal calcification, calculi of varying size and shape in the duct system, or both. Nuzum[17] in 1946 stated that in diffuse calcification of the pancreas the process of calcification is distributed throughout the organ, with associated destruction of the parenchyma. Eliason and Welty[11] in 1948 were of the opinion that many of the cases of diffuse calcification of the pancreas represent cases with hundreds of calculi, most of which will of necessity occupy the smallest ducts and, therefore, appear at first glance to lie within the substance of the gland itself. Wirts and Snape[27] in 1947 stated that the diffuse gritty sensation observed on cutting into the pancreas and the diffuse infiltration noted upon roentgen examination might be the result of very fine radicles of the pancreatic ducts being almost completely filled with deposits of finely granular calcium salts and suggested that the deposition of calcium might be simply a progressive phase of the condition causing the deposit of pancreatic calculi in the larger ducts.

On the basis of the histologic sections of the pancreas in 6 of our cases (3 autopsied and 3 operated), we are of the opinion that the diffuse calcification of the pancreas in these 6 cases represents an advanced stage of pancreatic lithiasis, with the so-called parenchymal calcification representing small amorphous appearing calculi in the fine radicles of the pancreatic ducts. These areas of calcification may appear to be in the parenchyma because the lining epithelium of the radicles becomes denuded, most likely due to pressure necrosis, infection and stasis (Fig. 1). The characteristic microscopic appearance of our cases of diffuse calcification of the pancreas is the replacement of most of the parenchyma by dense, acellular fibrous tissue, with marked dilatation and deformity of the ducts, absence of most of the acini, preservation of some of

TABLE I

RECORDED CASES IN THE LITERATURE OF DIFFUSE CALCIFICATION OF THE PANCREAS

Authors	Year	No. Cases
Smith and Bonis[23]	1941	3
Beling, Baker and Marquis[2]	1942	1
Darling[6]	1942	1
Pasternack[18]	1943	3
Whipple[25]	1943	3
Nuzum[17]	1946	1
Comfort, Gambill and Baggenstoss[5]	1946	3
Present and Geyman[20]	1947	2
Wirts and Snape[27] (Review of literature and 2 cases of their own.)	1947	24
Reeves and Moran[21]	1948	6
Epstein and Isaacs[12]	1948	1
Buckstein[4]	1948	4
Mourao and Schindler[16]	1949	1
Williams[26]	1949	1
Domzalski[7]	1949	2
Peters, Lubitz and Lindert[19]	1951	21
Total		77

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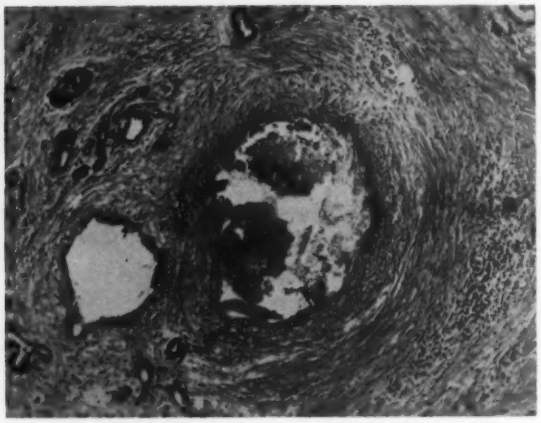

Fig. 1. Photomicrograph of biopsy of the pancreas showing amorphous appearing calcification in a small radicle of a pancreatic duct. Note the replacement of most of the parenchyma by fibrous tissue and the inflammatory round cell infiltration. (Hematoxylin and eosin, ×92.)

the islets of Langerhans and chronic inflammatory round cell infiltration. All of our 6 cases had calculi in the larger and smaller ducts (Fig. 2) and in none of them were we able to demonstrate any definite parenchymal calcification.[10]

This microscopic observation that the calculi in diffuse calcification of the pancreas are in the ducts and not the parenchyma corresponds to the recent conclusions of Peters, Lubitz and Lindert.[19] They studied 6 autopsy and 1 surgical specimen

Fig. 2. Photomicrograph of biopsy of the pancreas showing calculi in a pancreatic duct. (Hematoxylin and eosin, ×368.)

of diffuse calcification of the pancreas and found the calculi either in larger or finer duct radicles or in spaces without perceptible epithelial lining. From a pathologic standpoint free intraductal calcifications or stones and diffuse calcification of the pancreas would seem to be the same with the latter a progressive phase of the former.

ETIOLOGY

The etiology of diffuse calcification of the pancreas has not been definitely determined. Alcoholism is supposed to have some significance but this may represent an attempt on the part of the patient to obtain relief from pain rather than a cause for the calcification. Eleven of our patients gave a definite history of chronic alcoholism. Chronic disease of the biliary tract may have some significance but Haggard and Kirtley[13] found only 9 cases of biliary calculi in 56 cases of pancreolithiasis in which there was surgical intervention. None of our 6 autopsied or operated cases showed evidence of biliary disease.

The most likely cause of calcification in the pancreas is previous attacks of pancreatitis. Pancreatic calculi are known to be composed of calcium carbonate and tribasic calcium phosphate. Snell and Comfort[24] believed that normal pancreatic juice did not contain calcium in this form and it was probable that inflammatory processes in the pancreas were responsible for alternation of the chemical composition of pancreatic secretions and the subsequent deposition of calcium within ducts. Edmondson and Fields[9] have shown that in cases of acute pancreatitis a large amount of calcium is mobilized in the pancreas in the area of fat necrosis. The lipase in these areas of fat necrosis splits the neutral fat to fatty acids and glycerin and the fatty acids combine with calcium to form mostly insoluble soaps. Bockus[3] states that in this way calcification of the pancreatic parenchyma may take place in so-called diffuse calcification of the pancreas and similar calcium deposits in the duct walls might also

act as a nidus for the development of calculi in some instances.

However, the mobilization of calcium in areas of fat necrosis in acute pancreatitis does not account for the formation of stones within the ducts. Edmondson *et al.*[8] in a recent study of the physical chemistry involved in the formation of calculi found that under normal conditions the pancreatic juice is supersaturated with calcium carbonate and postulate that an attack of pancreatitis or stasis secondary to chronic inflammation may initiate the precipitation of calcium carbonate.

Assuming that pancreatitis is the cause for diffuse calcification of the pancreas raises the possibility of some relation to cystic fibrosis of the pancreas in childhood. Wirts and Snape[27] and Williams[26] have mentioned this in the literature. The microscopic picture in cystic fibrosis of the pancreas bears a striking similarity to that found in diffuse calcification, even to the extent of tiny calcified concretions. The clinical entity of cystic fibrosis of the pancreas in infancy and childhood has been divided into three groups: (a) those dying during the neonatal period of intestinal obstruction from inspissated meconium; (b) those dying between the neonatal period and six months of age with failure to gain weight; (c) those dying after six months with the celiac syndrome and pulmonary infection. It might be that many of these infants have a mild form of the disease and years later develop diffuse calcification of the pancreas, with or without symptoms. Patients with cystic fibrosis of the pancreas and those with diffuse calcification of the pancreas are both subject to complicating pulmonary infection.

Zuidema[28] recently reported 4 cases of diffuse calcification of the pancreas in patients whose age ranged from twenty to twenty-eight. All of these patients were Indonesians from central Java who had developed diabetes. None of them had a history of pancreatitis or abdominal pain and none had ever ingested alcohol. All the patients belonged to a very poor social class in which hepatic cirrhosis is common. Their diet was markedly deficient in all respects but particularly in animal protein and consisted largely of rice. He concluded that the cause of the change in the pancreas with the calcium salt disposition was due to protein deficiency, most likely amino acids.

DIAGNOSIS

The most important and easiest diagnostic procedure is roentgen examination of the abdomen. The characteristic appearance is that of diffuse calcium deposition outlining the position of the pancreas. The calcium deposits extend obliquely from just to the right of the first or second lumbar vertebrae upward to the left upper quadrant at the level of the eleventh or twelfth dorsal vertebrae (Fig. 3). In the lateral view of the abdomen the calcifications are seen just an-

Fig. 3. Anteroposterior roentgenogram showing diffuse calcification outlining the position of the pancreas. The calcification extends from the right of the second lumbar vertebra upward to the left upper quadrant to the level of the twelfth dorsal vertebra.

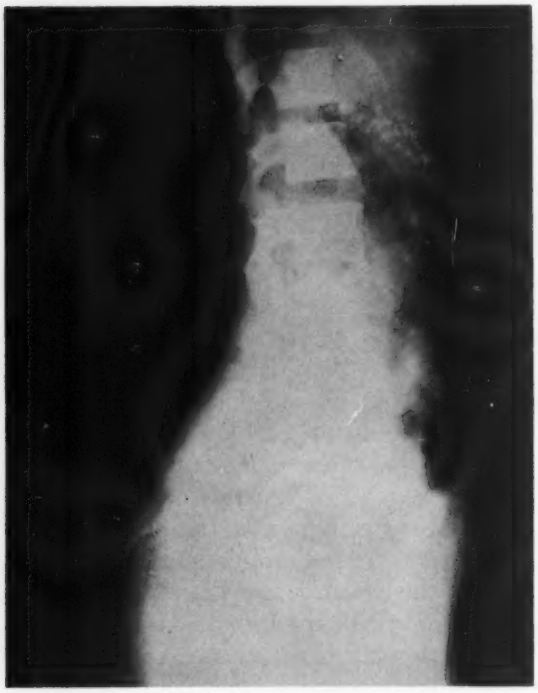

Fig. 4. Lateral roentgenogram. The calcification outlines the position of the pancreas just anterior to the bodies of the upper lumbar vertebrae.

Fig. 5. Intravenous pyelogram showing the relation of the pancreatic calcification to the kidneys visualized with opaque medium.

terior to the bodies of the vertebrae (Fig. 4). These calcific shadows must be differentiated from renal and biliary calculi, calcifications of costal cartilages, liver and spleen, calcified mesenteric lymph nodes and calcified plaques in the aorta and splenic vessels. The relation of the pancreatic calcification to the kidneys is shown in Figure 5 and to the transverse colon in Figure 6. With barium in the stomach the calcification is seen in the center of the duodenal loop and just above the lesser curvature of the stomach (Fig. 7).

Wirts and Snape[27] have stressed the importance of absent or diminished pancreatic secretion as determined by means of simultaneous aspiration of gastric and duodenal secretions after intravenous injection of secretin. This procedure was not carried out in any of our patients.

Stool examination may help in the diagnosis, particularly if steatorrhea is present. Free fat was found in the stools of 2 of our

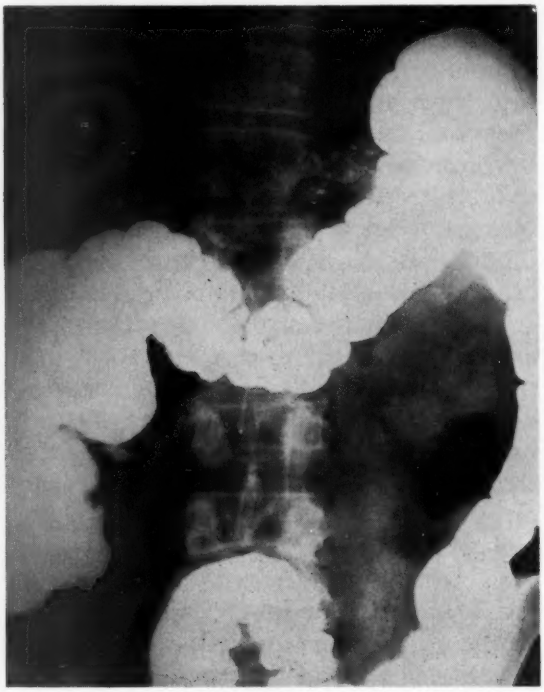

Fig. 6. Barium enema with the calcification of the pancreas just above the mid-transverse colon. The barium filled transverse colon may at times overlie the pancreatic calcification.

patients, occult blood in 5, 7 were recorded as normal and 4 had no record of any stool examination.

SYMPTOMS AND SIGNS

The average age of our patients was 46.2 years, the youngest being 32 years and the oldest 61 years. There were 12 males and 6 females; 10 being white and 8 colored.

The presenting symptoms were upper abdominal pain, vomiting and diarrhea. Abdominal pain was present in 15 cases; the pain was usually epigastric, either intermittent or constant, and in only 2 of the cases did the pain radiate to the back and left shoulder. Vomiting was present in 10 cases and diarrhea in 2.

The physical findings were those of abdominal epigastric tenderness in 12 cases, a palpable, firm, nodular mass just above and to the left of the umbilicus in 4 cases and evidence of weight loss in 8 cases.

It is interesting to note that some cases of diffuse calcification of the pancreas have no symptoms or signs referable to the pancreas. Two of our patients were in this category and the presence of the calcification was an incidental finding on roentgen examination of the abdomen. Neither of these patients had abdominal pain, steatorrhea or diabetes and there was nothing in the past history to suggest pancreatitis. It is possible that the calcium was deposited in the pancreas as a result of a low-grade, subclinical pancreatitis dating back to childhood.

It is to be remembered that the finding of pancreatic calcification does not always indicate pancreatitis. The roentgenologist should always report the presence of pancreatic calcification and then it is up to the clinician to determine if pancreatitis is the cause of the patient's symptoms. Unfortunately the roentgenologist cannot determine which cases of diffuse calcification of the pancreas have chronic pancreatitis but his report of calcification may be a strong lead in the differential diagnosis of a patient with upper abdominal pain.

Other causes for upper abdominal pain may be associated with diffuse calcification of the pancreas. Four of our patients had a roentgen diagnosis of duodenal ulcer at one time during their repeated hospital admissions.

Fig. 7. Calcification in the head of the pancreas is seen in the center of the duodenal loop and the calcification of the body just above the lesser curvature of the antral portion of the stomach.

Mental symptoms may cloud the diagnosis particularly when associated with the persistent upper abdominal pain, for which no reason has been found. Two of our patients had admissions to the psychopathic wards with a diagnosis of constitutional psychopathic state.

LABORATORY STUDIES

The serum amylase was determined in 7 cases and was elevated in 6 of these, ranging from 128 to 348 units (normal 25–125 units, average 60 units). The serum lipase was elevated in 2 cases, was normal in 2 cases and was not done in the other 14 cases. Serum total protein studies were carried out in 14 cases, the lowest being 5.5

grams/100 cc. and the highest 8.6 grams/100 cc. with the albumin globulin ratio reversed in 3 cases. The Wassermann reaction was positive in 1 case and negative in 17. The white blood cell count was elevated in only 4 cases, highest count being 17,700. Albumin studies of the urine were positive in 7 cases and negative in 10. The urine of 8 cases was negative for sugar and positive in 10, with 6 of these being definite diabetics by fasting blood sugar studies and glucose tolerance curves.

Because of the high incidence of diabetes in patients with diffuse calcification of the pancreas, it was thought that additional cases of pancreatic calcification would be found by roentgen examination of the abdomen of diabetics. Accordingly 112 patients from the diabetic clinic were examined roentgenographically but no new cases of diffuse pancreatic calcification were found.

Electrocardiographic studies were done in 7 cases, 6 of which were normal and one revealed evidence of coronary artery disease.

Of the above laboratory studies the serum amylase is the most significant. The amylase studies were all done within several days after admission and the elevated findings in 6 cases we believe indicate an associated pancreatitis.

TREATMENT

The immediate treatment for most of these patients is relief of pain. In severe cases morphine is necessary. One of our patients, treated for intermittent severe pain over a period of seven years, was a definite morphine addict. If the pain is intolerable and persistent, celiac ganglionectomy would seem to be the next step,[21] followed by partial or total pancreatectomy if necessary[25]

Reinhoff and Baker[22] report a case in which a bilateral sympathectomy from the fifth thoracic to the second lumbar ganglion, together with a transthoracic bilateral vagectomy provided complete relief of pain and changed a chronic morphine addict of years' standing with a "psychopathic personality" back into a useful citizen. Recently transduodenal sphincteroplasty[14] combined with vagotomy has been advocated in the treatment of chronic recurrent pancreatitis. This procedure may be of some value in the relief of pain.

Surgical removal of the distal two-thirds of the pancreas was performed in 2 of our cases, one of whom later died of metastasis from carcinoma of the pancreas. To prevent the serious end results of pancreatic insufficiency and in those cases in which steatorrhea, weakness and weight loss are present, the advocated treatment is a high caloric, high protein, low fat, low residue diet and up to 10–30 grams daily of pancreatin in the form of enteric coated tablets along with massive doses of vitamins.

INCIDENCE OF TUBERCULOSIS

Ackman and Ross[1] reviewed 54 cases of proved pancreatic lithiasis, 29 of which were surgical and 25 autopsied cases. Of the 25 autopsied cases 6 had pulmonary tuberculosis. They reported their own case which at autopsy also showed tuberculosis. In this review 7 or 26.9 per cent of the autopsied cases of pancreatic lithiasis exhibited pulmonary tuberculosis.

Eight of our 18 cases are known to be dead. The cause of death in one case was carcinoma of the esophagus and in another was carcinoma of the pancreas. Six persons died of pulmonary tuberculosis. Four of these patients were white and 2 colored, which would indicate that the race was not a factor. Thus, 6 or 33.3 per cent of our cases have had complicating pulmonary tuberculosis as the cause of death.

A review of 77 cases of diffuse calcification of the pancreas described in the literature previous to this report shows that 19 deaths are recorded. Nine of these were due to pulmonary tuberculosis, 2 patients died of pulmonary suppuration, 1 of carcinoma of the pancreas, 1 of myocardial infarction, 1 of pancreatitis and abdominal abscess, 1 of bile peritonitis and the cause of the death in the other 4 was not recorded. As stated above 8 of our patients have died, 6 due to pulmonary tuberculosis. Thus, of the 27 re-

corded deaths in patients with diffuse calcification of the pancreas, 15 or 55.5 per cent were due to pulmonary tuberculosis.

Pasternack[18] states that patients with prolonged pancreatic insufficiency are predisposed to pulmonary tuberculosis, suppuration and gangrene. Others[3,11] claim that the tuberculosis is probably the result of lowered resistance or secondary to the extensive weight loss. The presence of pulmonary tuberculosis in 6 of our cases and the incidence in other cases as recorded above is probably more than coincidental. There is no evidence that tuberculosis is responsible for calcified areas in the pancreas but one of our cases was found to have tuberculous capsulitis of the liver and fibrocaseous tuberculous lymphadenitis of abdominal lymph nodes by biopsy at abdominal exploration six years before calcification of the pancreas was seen on roentgen examination. Extensive generalized tuberculosis seldom involves the pancreas, a fact for which no explanation is known.

Diffuse calcification of the pancreas particularly when associated with pancreatic insufficiency seems to predispose the patient to pulmonary infection. Repeated attacks of pneumonia are often elicited in the history of these patients, and in many cases are the cause of numerous admissions to the hospital. The question of vitamin A deficiency due to steatorrhea as a factor in the etiology has been mentioned in the literature. The microscopic examination of the lungs in our cases, however, revealed nothing unusual in the lining epithelium. This susceptibility to pulmonary infection seems to be similar to that seen in cystic fibrosis of the pancreas. When a patient with diffuse calcification of the pancreas developes tuberculosis, the ultimate outcome seems to be poor. All 6 of our patients with tuberculosis failed to respond to the usual therapy and died of the disease. Most of these patients were treated without the use of the new chemotherapeutic drugs and that factor may alter the prognosis.

The high incidence of pulmonary tuberculosis in patients with diffuse calcification of the pancreas, where the pancreas shows a marked fibrosis replacement of the acini, is unexplained. This would seem to be worthy of further investigation.

SUMMARY

1. Eighteen cases of diffuse calcification of the pancreas are reviewed. A search of the literature reveals 77 cases previous to this report.

2. The histologic sections of the pancreas in 6 of these cases revealed calculi in the ductal system and no evidence of calcification of the parenchyma. This finding, we believe, indicates that diffuse calcification of the pancreas represents just a progressive or advanced stage of pancreatic lithiasis.

3. The etiology of diffuse calcification of the pancreas is not definite, but the most likely cause is previous attacks of pancreatitis. Mild forms of cystic fibrosis of the pancreas in infancy may eventually develop diffuse calcification of the pancreas. A diet deficient in protein (amino acid) may have some etiological significance.

4. The most important and easiest diagnostic procedure is roentgen examination of the abdomen.

5. The most common symptoms are recurrent upper abdominal pain, vomiting, weight loss and diabetes. However, 2 of our patients had none of these symptoms and the calcification was diagnosed as an incidental finding in roentgen examination of the abdomen.

6. The serum amylase is the most significant laboratory study. In 6 of our cases the serum amylase was elevated indicating an associated pancreatitis.

7. Treatment includes the control of pancreatic insufficiency and pain. When the pain is intolerable severance of the autonomic fibers supplying the pancreas may help or, if necessary, partial or complete pancreatectomy should be performed. Sphincterotomy and cholecystectomy may be of value.

8. Pulmonary tuberculosis was the cause of death in 6 or 33.3 per cent of our cases.

Of the 27 recorded deaths in patients with diffuse calcification of the pancreas 15 or 55.5 per cent were due to tuberculosis. This high incidence remains unexplained.

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ADDENDUM

Sixteen more cases of diffuse calcification of the pancreas have appeared in the literature since this paper was written.

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VOL. 78, NO. 4

SIMULATION OF COLONIC OBSTRUCTION AT THE SPLENIC FLEXURE BY PANCREATITIS: ROENTGEN FEATURES*

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SEGMENTAL gas distention of the transverse colon is a well known phenomenon in pancreatitis. That the roentgenographic picture may simulate large bowel obstruction with areas of narrowing in or near the splenic flexure is not so well appreciated.

Forlini,[7] Baylin and Weeks,[2] Glenn and Baylin,[9] Remington, Mayo and Dockerty,[19] Miln and Barclay,[14] and Rose[21] presented a total of 8 cases of narrowing or obstruction of the colon secondary to pancreatitis. A review of these reports indicates that the adjacent inflammatory process may produce colonic obstruction in two ways: by inducing intermittent or constant spasm in the acute cases, or by colonic fibrosis in the chronic cases. Inflammatory masses incorporating the splenic flexure have been found in some cases, but it seems that spasm is responsible for obstruction more often than pressure by the mass, since, when the colon is dissected free, no remarkable narrowing can be demonstrated. In any event, such obstruction may be temporary, intermittent or persistent in duration, partial or complete in degree, and of a magnitude sufficient to require cecostomy.

The following three cases are examples of such colonic disturbance.

REPORT OF CASES

CASE I. J.C. PH #07704. This sixty-eight year old white male was admitted on April 26, 1953 with a chief complaint of abdominal pain of forty-eight hours' duration. Past history was not contributory. Because of questionable spasm and minimal tenderness in the right upper quadrant following an acute episode of postprandial abdominal pain and vomiting, a presumptive diagnosis of cholecystitis was made two days prior to admission. On the day before admission, the patient complained of back pain, had abdominal distention, a temperature of 100.4°F. and was icteric. On admission, there were scleral icterus, rales at the left base, and no abdominal pain, tenderness, distention, or palpable masses. Significant laboratory findings were: white blood cell count, 10,700; polynuclears, 92 per cent; alkaline phosphatase, 6.5 Bodansky units; bilirubin, 4.0 mg. per cent; erythrocyte sedimentation rate, 60. Plain film of the abdomen on April 27, 1953, revealed colonic distention up to the splenic flexure without gas distally. The roentgenographic impression was an obstructing lesion at the splenic flexure. During the next few days, the white blood cell count rose slowly, the icterus disappeared, and the abdomen became increasingly distended. Nevertheless, gas was passed per rectum and active peristalsis was heard. The patient remained febrile. A repeat roentgenogram of the abdomen on May 1, 1953, (Fig. 1) disclosed increasing distention of the large bowel, although some gas was now seen in the descending colon. A barium enema on May 4, 1953, demonstrated flattening of the medial wall of the proximal descending colon (Fig. 2). A repeat barium enema examination two days later showed the mucous membrane in the previously demonstrated area of flattening now to be edematous (Fig. 3). The plan of management was first to combat the chronic recurrent ileus with cecostomy, and then to proceed to definitive gallbladder surgery. Following cecostomy on May 8, 1953, the white blood cell count continued to rise and fever persisted.

At laparotomy on May 22, 1953, a cholecystectomy and choledochostomy were done for cholecystitis and cholelithiasis. The head and neck of the pancreas were inflamed and edematous, and contained many areas of fat necrosis.

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Fig. 1. Case 1. Pancreatitis with fat necrosis associated with cholecystitis and gallstones. A plain roentgenogram of the abdomen discloses gaseous distention of the colon proximal to the splenic flexure. The cecum is greatly distended. Distal to the splenic flexure, a small amount of gas is present.

Fig. 2. Case 1. A barium enema study three days after the roentgenogram shown in Figure 1 again reveals distention of the colon proximal to the splenic flexure. No evidence of obstruction was found. The medial wall of the proximal third of the descending colon is flattened.

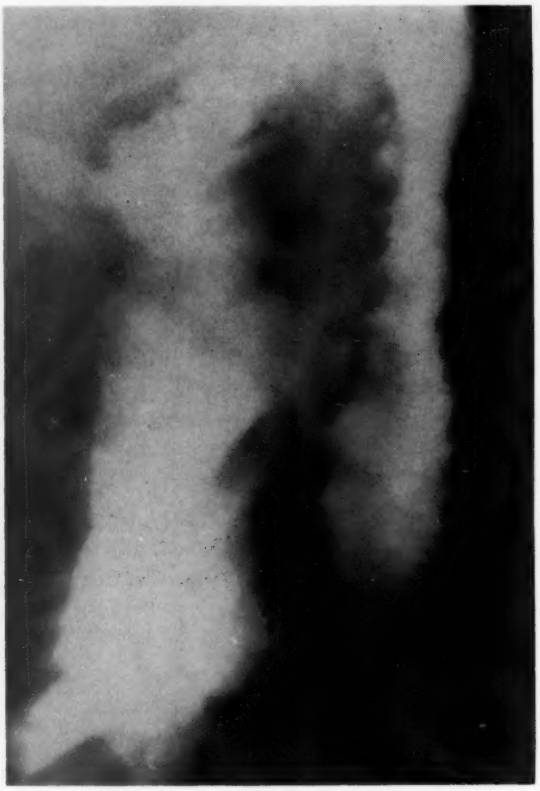

Fig. 3. Case 1. Enlargement of a second barium enema study image two days after the first study demonstrates widening and irregularity of the mucosal folds along the medial side of the descending colon which is consistent with edema of the mucosa.

The body and tail of the pancreas were slightly enlarged and firm. The transverse colon and splenic flexure were unremarkable on palpation.

Thirteen months later the patient was well. The final diagnoses were: acute and chronic cholecystitis, cholelithiasis, acute pancreatitis and fat necrosis.

Case II. J.L. PH #963388. This seventy-six year old white male was admitted on October 13, 1953, with chief complaints of abdominal distention, vomiting and crampy peri-umbilical pain of thirty hours' duration. There was no constipation.

The past history included surgery in January 1953, for cholecystitis and cholelithiasis. At this operation the head of the pancreas was found to be thickened, suggesting an inflammatory re-

action. The ascending and proximal transverse colon was normal. The splenic flexure and tail of the pancreas were not palpated. The postoperative course was not unusual.

On physical examination, the temperature was 100.4°F. and the abdomen was distended. There were peri-umbilical tenderness and hyperactive bowel sounds. The clinical impression was intestinal obstruction. Laboratory findings were: white blood cell count, 14,000; polynuclears, 88 per cent; serum amylase, 148 Myers-Killian units (normal 10–35).

A plain roentgenogram of the abdomen (Fig. 4) showed moderate distention of the small bowel and colon reaching to the splenic flexure with no gas distally. The impression was obstruction at the splenic flexure. During an emergency barium enema examination, fluoroscopy revealed an area of narrowing at the splenic flexure, which disappeared after several minutes (Fig. 5). The impression was severe spasm at the splenic flexure secondary to an inflammatory intra-abdominal process. During the next two weeks, the patient continued to run a low grade fever and the white blood cell count remained elevated. The serum amyllase became normal. Treatment with Miller-Abbott tube drainage and antibiotics reduced abdominal pain but distention persisted.

Fig. 4. Case 11. Abscess of the body of the pancreas. A scout roentgenogram of the abdomen shows gaseous distention of the colon proximal to the splenic flexure.

Fig. 5. Case 11. An oblique roentgenogram taken during a barium enema examination performed the same day as in Figure 4 discloses an apparently normal splenic flexure. At fluoroscopy, spasm of the splenic flexure was present but subsided after several minutes.

On October 29, 1953, a repeat barium enema examination demonstrated a persistent area of narrowing in the proximal descending colon with irregularity of the medial wall just distal to the constriction (Fig. 6). On the postevacuation film, the barium proximal to the constriction was retained. Opinions as to causation varied between a small carcinoma with associated spasm and an inflammatory process with central ulceration, such as tuberculosis. It was agreed, however, that exploration was warranted.

On October 30, 1953, a preliminary cecostomy was performed, and on November 11, 1953, at exploratory laparotomy, a 6 by 8 by 7 cm. abscess of the body of the pancreas was found. There was definite inflammatory reaction of the transverse mesocolon, with foreshortening of this structure. The process also extended toward the spleen, which was adherent to the diaphragm and to the posterior parietal peritoneum. The gastrocolic ligament was slightly edematous. The transverse colon and splenic flexure were normal to palpation. It was the surgeon's impression that the area

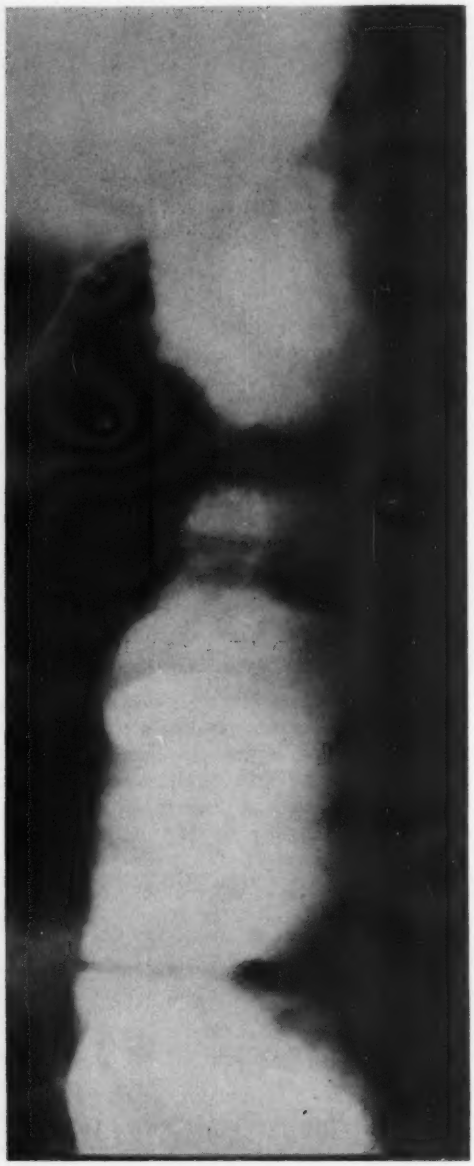

Fig. 6. Case II. Enlargement of a second barium enema study image sixteen days after the first study reveals a constant narrowing of the proximal descending colon, and irregularity of the medial wall just distal to the constriction.

of narrowing in question had been produced by spasm.

The patient was well ten months later. The final diagnosis was pancreatic abscess.

Case III. J.H. #392067. This thirty year old colored female, a chronic alcoholic, was admitted on March 17, 1955, with a chief complaint of back pain of three days' duration following overindulgence in alcohol. Successively, there appeared abdominal distention, excessive flatus, eructations, anorexia, nausea and vomiting. There had been no bowel movement for two days.

On physical examination the patient was in moderate distress. The abdomen was distended, with diffuse rebound tenderness most marked in the upper quadrants. Bowel sounds were hypoactive. The temperature was 100.4°F. Laboratory findings were: white blood cell count, 13,500; serum amylase, 395 Somogyi units with a control of 40. The clinical impression was acute pancreatitis.

A plain roentgenogram of the abdomen on admission showed moderate distention of the colon proximal to the splenic flexure with no gas distally. The distance between the stomach and the transverse colon appeared increased (Fig. 7). The impression was obstruction at the splenic flexure. During an emergency barium enema examination, fluoroscopy disclosed spasm just proximal to the splenic flexure, which finally subsided. Unfortunately, no spot roentgenogram was taken. On the postevacuation film an area of narrowing was seen in the same location (Fig. 8). The impression

Fig. 7. Case III. Recurrent acute pancreatitis. A plain roentgenogram of the abdomen demonstrates gaseous distention of the colon proximal to the splenic flexure and absence of gas distal to this point. The cecum is markedly dilated. The distance between the stomach and transverse colon appears to be increased.

Fig. 8. Case III. A postevacuation roentgenogram of a barium enema examination done the same day as in Figure 7 reveals the inconstant area of narrowing just proximal to the splenic flexure, which was seen at fluoroscopy.

was pancreatic abscess draining into the transverse mesocolon.

Occasionally, the patient's temperature rose to 103°F. On conservative treatment, she gradually improved and was discharged afebrile on March 25, 1955.

The patient was re-admitted on July 11, 1955, with generalized severe crampy abdominal pain again following alcoholic excess. Other symptoms were similar to those on the first admission.

On physical examination the temperature was 104.8°F. The abdomen was distended, with generalized tenderness and hypoactive bowel sounds. The laboratory findings were: white blood cell count, 23,800; amylase, 300 Somogyi units with a control of 175. The disease ran an irregular course with exacerbations and remissions.

At laparotomy on July 22, 1955, the entire pancreas was enlarged, edematous and hard, and an area of necrosis was found in the head of the pancreas. The colon was not remarkable. Following a stormy course, the patient was discharged much improved, but interval exacerbation has since occurred. The final diagnosis was chronic and acute pancreatitis.

DISCUSSION OF ROENTGENOGRAPHIC FINDINGS

In the case of Baylin and Weeks,[2] barium enema examination revealed three areas of disturbance which they attributed to adjacent fat necrosis. The transverse colon and the splenic flexure showed zones of persistent spasm and mucosal alteration; the cecum and terminal ileum were narrowed. Glenn and Baylin[9] presented a single twenty-four hour film, presumably made after ingestion of barium, demonstrating an area of narrowing in the proximal transverse colon which they attributed to spasm. In the first case of Remington, Mayo, and Dockerty,[19] an area of narrowing in the splenic flexure was interpreted as carcinoma. Their second case disclosed an area of narrowing at the splenic flexure "without mucosal changes characteristic of carcinoma" and a diagnosis of an "indeterminate lesion" was made. We, however, feel that the published roentgenogram is most suggestive of carcinoma. Roentgenograms were not published in the remaining cases to which reference is made.

In the literature[1,4,8,13,16] illustrations and descriptions indicate that distention of the transverse colon in pancreatitis has been observed by itself or in association with various states of distention of other portions of the intestine as part of a generalized process of abnormal distention involving both small and large bowel; accompanied by distended loops of small intestine only; as a localized phenomenon without concomitant dilatation of the right colon or else with associated cecal distention. In the last instance, however, we have found no special comment made about the significance of the cecal change. Bockus *et al.*[3] mention 4 cases in which plain roentgenograms of the abdomen showed findings compatible with mechanical or paralytic ileus. No roentgenograms were reproduced in the article. Presumably, they mean that gas was present throughout the intestinal tract.

Our 3 cases are strikingly similar. Ini-

tially, they all showed abnormal distention of the colon from the cecum to the splenic flexure. This is the roentgenographic picture of obstruction, not segmental gas distention. The rather marked degree of cecal distention in 2 of our cases (Cases I and III) emphasizes this point. In all 3 cases, the initial impression after study of the plain roentgenogram of the abdomen was obstruction at the splenic flexure.

Our barium enema findings were: (a) an area of persistent spasm in the proximal descending colon seen on two separate occasions (Case I); (b) an area of intermittent spasm just proximal to the splenic flexure (Case III); (c) an area of temporary spasm at the splenic flexure, changing at a later examination to (d) an area of persistent narrowing of the proximal descending colon with irregularity of the medial wall just distal to the constriction (Case II).

Poppel[17] states that "in the earlier stages of acute pancreatitis, there is spasm and irritability of the mid-transverse colon, later on involving the left half and finally the entire transverse colon and splenic flexure. This is accordingly manifested by intolerance to barium. Later, the mucosa may become edematous and produce the indenture pattern indicating protrusion of edematous folds of mucous membrane into the lumen with narrowing of the lumen."

A study of the operative or autopsy findings in the cases of Forlini,[7] of Miln and Barclay,[14] and of Rose[21] makes it clear that plain roentgenograms of the abdomen, had they been made, would have exactly resembled those of our cases. Distention of the colon is not a prominent feature of the other cases to which we have made reference.

DISCUSSION

Ten of the 11 cases discussed showed areas of spasm manifested by narrowing at or near the splenic flexure. Why this predilection should occur is not known. There is no indication that pancreatitis is commonly restricted to the tail, and such involvement was certainly not present in our 3 cases. In other words, distention of the colon proximal to the splenic flexure has been found regardless of whether or not the tail of the pancreas was involved.

A brief review of certain anatomic relations of the pancreas may be helpful in considering possible avenues of spread of inflammatory exudate. The greater part of the right half of the anterior surface of the head of the pancreas is in contact with the transverse colon. The left extremity of the inferior surface of the body rests on the splenic flexure, which is also in contact with the tail, since the latter lies in the phrenolienal ligament.[5,10,15]

The peritoneal reflections (Fig. 9) are such that one may anticipate involvement of practically any segment of the alimentary tract. The transverse mesocolon offers a direct route to the transverse colon; the phrenocolic ligament to the splenic flexure.

According to Evans and Ochsner[6] and Rouvière[22] the pancreatic lymphatic collecting trunks drain into several richly an-

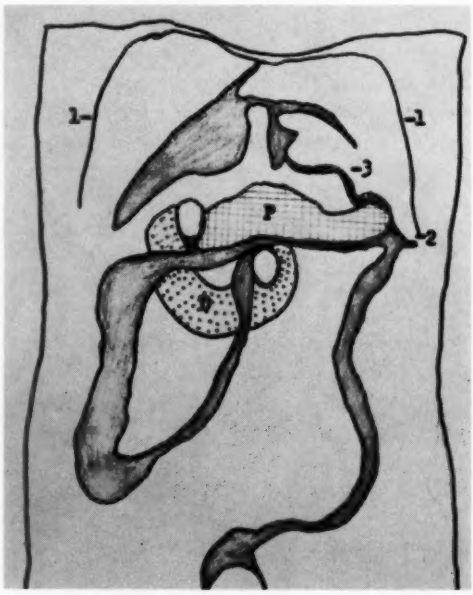

Fig. 9. The shaded areas represent the peritoneal reflections on the posterior abdominal wall.

P Pancreas
D Duodenum
1. Diaphragm
2. Phrenocolic Ligament
3. Gastrolienal Ligament

astomosing groups of lymph nodes. Of particular interest to us are the lymph nodes in the phrenolienal ligament, which receive some of the drainage from the tail, and the lymph nodes at the root of the mesentery, which receive some of the drainage from the body and head.

The roentgenographic demonstration of narrowing at the splenic flexure and proximal colonic distention can occur long before organic changes in bowel wall appear. The cases of Remington *et al.*[19] were chronic and did show fibrosis of the muscularis of the colon. On the other hand, in our cases and in those of Miln and Barclay[14] and of Rose,[21] the colon was grossly normal at operation and devoid of indication for colectomy. Further, in our 3 cases, the patients' subsequent courses did not suggest continued bowel obstruction, as might be expected in the face of organic bowel change.

Some workers feel that distention is secondary to contact with escaping pancreatic enzymes.[11,13] This does not suffice to explain the striking localization of the constriction at the splenic flexure, unless, for some reason, the exudate collects only at that point or initiates a visceral reflex.

Data are available on the status of the splenic flexure in 9 cases. In 6 there was definite evidence of an inflammatory or fibrotic process close to or involving the splenic flexure, and adequate to account for the narrowing there. A very similar explanation may exist in the other cases. Figure 9 illustrates how inflammatory exudate can spread from the pancreas along the phrenocolic ligament to the vicinity of the splenic flexure. One may then postulate secondary spasm resulting in colon obstruction, with small bowel distention ensuing in those cases where the ileocecal valve is incompetent.

Another possible explanation for colonic distention up to, but not beyond, the splenic flexure is a visceral reflex. The colon, to this point, receives its autonomic supply from the superior mesenteric plexus[18] the branches of which accompany those of the superior mesenteric artery. Any reflex that results in relative or absolute sympathatonia could explain this distribution of distention. Further, inflammation of the lymph nodes in the root of the mesentery may account for cases where both small and large bowel distention is found, whereas inflammatory exudate confined to the transverse mesocolon may explain solitary distention of the transverse colon.

Arguing in favor of such a concept are the cases of superior mesenteric vascular thrombosis reported by Rendich and Harrington[20] and Harrington.[12] The plain roentgenograms of the abdomen in their cases are quite similar to ours. What is more remarkable is that in 4 of their 7 cases no gangrene or discoloration of any part of the colon was mentioned, although in all 7 the colon was abnormally dilated up to the splenic flexure. Further, in their 3 survivors, only small bowel resections were performed.

A visceral reflex of the sort postulated explains why mesenteric thrombosis and pancreatitis, two obviously different morbid processes, may sometimes produce an almost identical roentgen appearance. This hypothesis does not satisfactorily explain intermittent or persistent localized spasm, mucosal edema, and irregularity of the bowel wall demonstrated in our and in other cases.

DIFFERENTIAL DIAGNOSIS

Segmental gas distention may occur in relation to any acute intra-abdominal inflammatory process. For the reasons already given, the distention we discuss does not appear to be of this type.

Primary inflammations of the colon, in atypical location at the splenic flexure, such as tuberculosis, amebiasis or diverticulitis, may produce localized narrowing and proximal distention. Inconstant narrowing, by itself, such as appeared in one of our cases of pancreatitis, would argue against primary colonic disease. Adhesions secondary to peritonitis or previous surgery may narrow the splenic flexure (Fig. 10).

Fig. 10. J.H. #381925. This sixty-five year old white female had crampy abdominal pain for forty-eight hours. A barium enema study shows a constant area of narrowing in the transverse colon just proximal to the splenic flexure. At operation, dense adhesions were found, binding the colon to the spleen. No intrinsic disease was present on colotomy.

In pancreatitis, persistent narrowing capable of obliterating the mucosal pattern of the splenic flexure or sufficient mucosal edema to produce obstruction may well simulate carcinoma, especially when the colon is distended proximally and collapsed distally. Relative youth, as in Case III, sometimes points away from neoplasia.

The roentgen findings in superior mesenteric vascular thrombosis may be very similar, but distention of the small bowel would appear to be more prominent than in our cases.

There is probably no typical roentgen appearance. Nonetheless, distention of the colon proximal to the splenic flexure with a persistent or intermittent area of narrowing without definite destruction of mucous membrane may be regarded as consistent with pancreatitis. The clinical findings—alcoholism, cholecystitis, icterus, back pain, shock-like state, nausea and vomiting, abdominal tenderness, fever, elevated white blood cell count and amylase—may be helpful. Unfortunately, the clinical features of mesenteric thrombosis and pancreatitis may be similar.

Although the course of the disease was acute or subacute in each of our 3 cases, a similar roentgen appearance may be produced by chronic pancreatitis.[19]

We have seen 5 additional cases of suspected pancreatitis with distention of the colon proximal to the splenic flexure. None of these cases was surgically confirmed. In 4 of them the clinical diagnosis was pancreatitis. Two had a normal serum amylase and a negative barium enema study. Two others had marked elevation of serum amylase and were too ill to have a barium enema examination (Fig. 11 and 12). In the

Fig. 11. J.H. #401026. This fifty-nine year old white female had colicky abdominal pain, nausea and vomiting of nine hours' duration. Her temperature was 102°F., white blood cell count, 18,000 and serum amylase 900 Somogyi units. Plain roentgenogram of the abdomen shows distention of both small and large bowel with no colonic gas distal to the splenic flexure.

Fig. 12. J.H. #400961. This thirty-one year old colored male had abdominal pain and rigidity, nausea and vomiting for twenty-four hours. His temperature was 101.6°F., white blood cell count, 10,850 and serum amylase 450 Somogyi units. Plain roentgenogram of the abdomen shows moderate colonic distention up to the splenic flexure with no gas distally.

fifth, the clinical diagnosis was left renal colic, but no calculus could be demonstrated. No barium enema examination was performed. A gastrointestinal series showed calcification in the head and body of the pancreas, widening of the duodenal loop, and extrinsic pressure on the medial wall of the descending limb of the duodenum.

SUMMARY

As illustrated by 3 cases, the roentgenographic and fluoroscopic appearance of persistent or intermittent narrowing at or near the splenic flexure, in association with abnormal distention of the colon proximal to that point, with or without accompanying small intestinal distention is consistent with pancreatitis. In the 8 cases of this disease showing colonic narrowing, which have been recorded in the literature, the splenic flexure was the most commonly narrowed area.

A completely adequate explanation for this predilection is not known. The best hypothesis is that the inflammatory exudate spreads from the pancreas along the phrenocolic ligament to the vicinity of the splenic flexure where it produces spasm and consequent dilatation of the proximal colon and, in a patient with an incompetent ileocecal valve, small bowel distention as well.

Differentiation must be made from carcinoma of the splenic flexure, superior mesenteric vascular thrombosis, adhesions, and primary inflammations of the splenic flexure such as tuberculosis, amebiasis and diverticulitis.

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THE NONVISUALIZING AND POORLY VISUALIZING GALLBLADDER

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IN THE daily practice of radiology in a 300 bed, private hospital with a large volume of gastrointestinal work, the author has been impressed by the lack of understanding and confusion in the minds of the referring physicians when they are confronted by a report of a nonvisualizing or poorly visualizing gallbladder. The immediate reaction of the physician is to assume that the gallbladder does not function or that it functions poorly. Yet the failure to visualize may be due to faulty absorption from the intestinal tract or to liver disease or rarely to congenital absence of the viscus.

While Kirklin[6] concluded that the absent and faint shadows are "reliable indicia of cholecystic disease," Holmes, as quoted by Sosman,[13] observed that "variations in density of the gallbladder shadow are more often due to faulty technique than to disease." Statistical studies abound in the literature correlating the roentgen and operative findings. Revealing is that of Hodges and Lampe,[4] who reported that of 121 patients operated upon with the diagnosis of nonvisualizing gallbladders, 20 per cent were normal; and of 23 patients with faint shadows, only 61 per cent had significant gallbladder disease at surgery. Others[1,5,10,14] report better percentages, but, as Brewer[2] points out, such studies are misleading because the cholecystogram is not (and should not be) the sole basis of the selection of patients for surgery, and he asserts that the nonvisualization of the *normal* gallbladder is probably more frequent than most papers concede.

Since the surgeon and his medical colleagues are prone to rely on the radiologist's opinion it behooves him to choose his words carefully so as to prevent needless medical intervention or unnecessary surgery for nonexistent disease. Indeed, some surgeons[1,8,14] have long been wary of the poor or absent shadow and have advised caution in operating upon such evidence. The ensuing discussion is presented to again call attention to the undesirability of interpreting the cholecystogram in terms of gallbladder function, lest his professional colleagues be misled.

The roentgen examination of the gallbladder has reached a high state of perfection since the inception of cholecystography by Graham in 1924. The introduction of new media and the employment of "gravity" procedures, as popularized by Kirklin and others, have made possible easy identification of stones in the visualizing gallbladder.

The absent or faint shadow of gallbladder, incident to cholecystography, presents a somewhat different situation. Brief consideration of the anatomic and physiologic mechanisms involved in the opacification of the gallbladder during cholecystography should clarify this issue and demonstrate the fallacy of identifying a nonopacifying gallbladder as a nonfunctioning organ. Opacification by the oral method requires that a *sufficient quantity of contrast agent* be ingested, satisfactorily make the transit from mouth to stomach, pass the pylorus, remain sufficiently long in small bowel and be absorbed through the mucosa of small intestine, travel via portal circulation to liver, traverse the liver parenchyma, be excreted into the bile canaliculi, descend the biliary tree to cystic duct, enter and ascend cystic duct, and gain admission to the gallbladder. If this circuitous and hazardous route has been successfully navigated by *sufficient quantity* of the opaque medium, then the gallbladder, if properly roentgenographed after the appropriate interval has elapsed, may demonstrate its capacity to concentrate the iodinated com-

pound so that its shadow will be recorded on a cholecystogram. If there is any break in this long and complicated pathway, partially or wholly inadequate amounts of the contrast medium will reach the gallbladder and its roentgen demonstration may fail completely, or only a pale image may be recorded, depending on the extent of the interruption.

Visualization thus depends upon a patent channel from mouth to gallbladder, adequate absorption from bowel, satisfactory liver function and a properly functioning gallbladder. Proper operation of one further mechanism is essential: the sphincter of Oddi must be closed in order that the bile transporting the contrast agent shall enter the gallbladder and not flow unimpeded into the duodenum. Goulston[3] has emphasized this and has noted that the tone of the sphincter is increased during fasting and if alkali is present in the stomach.

One other factor is assumed to produce possible interference: physiologic stasis. Brewer,[2] Goulston[3] and others believe that if the patient has been fasting or on a low fat intake for a prolonged period of time, the gallbladder may be filled with a thick, concentrated bile which might prevent the entrance of the fresh, dye-laden bile.

Since the visualization of the gallbladder is thus seen to be dependent upon the successful accomplishment of a whole series of events—psychologic, anatomic, physiologic and roentgenographic—near and remote from this organ, it would appear unwise to assay the result of cholecystography in terms of gallbladder function.

Nor must one rely solely upon theoretic considerations to reach this conclusion, for frequent are the situations in practice where the poor or absent shadow is caused by extraneous factors, not by a diseased gallbladder. It is this experience which has prompted this communication. During a relatively brief span, patients submitting to cholecystography presented an interesting array of "pre-cholecystographic" pathologic states responsible for poor or absent visualization. They ran the gamut from the patient who disposed of his telepaque tablets in the toilet, to the one who left them on the bedside table, to the occasional patient who vomited promptly and persistently after ingestion of the medium or who took his tablets at the end of his night shift, shortly prior to reporting to the radiologic department, rather than the previous evening. They included patients with obstructing ulcers and neoplasms of the stomach, patients with gastric atony without demonstrable gastric or duodenal lesions, some in whom the contrast agent appeared to traverse the intestine unabsorbed, and patients with extensive liver disease due to various causes. Encountered also were patients who had failed to disclose to their referring physician that they had previously had a cholecystectomy or whose physician had forgotten this essential bit of information when ordering the work-up.

The literature on the nonvisualizing gallbladder is replete with at least passing mention of the need for considering all the foregoing factors[3,10,12] and Martin and

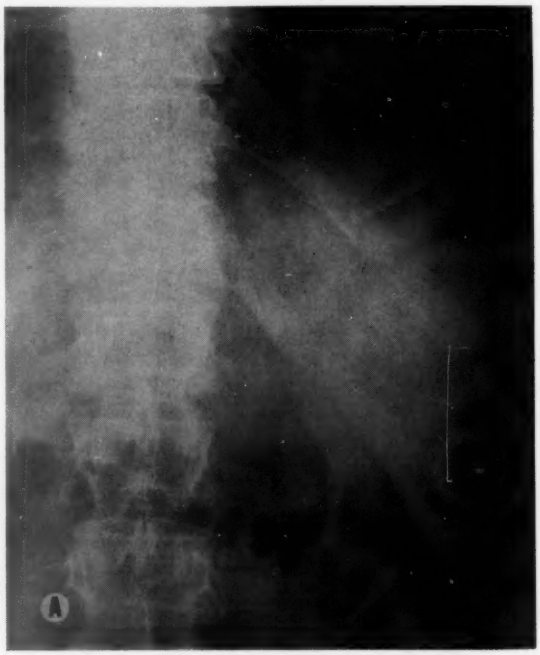

Fig. 1. Case 1. (*A*) Preliminary roentgenogram of gallbladder area (October 6, 1953).

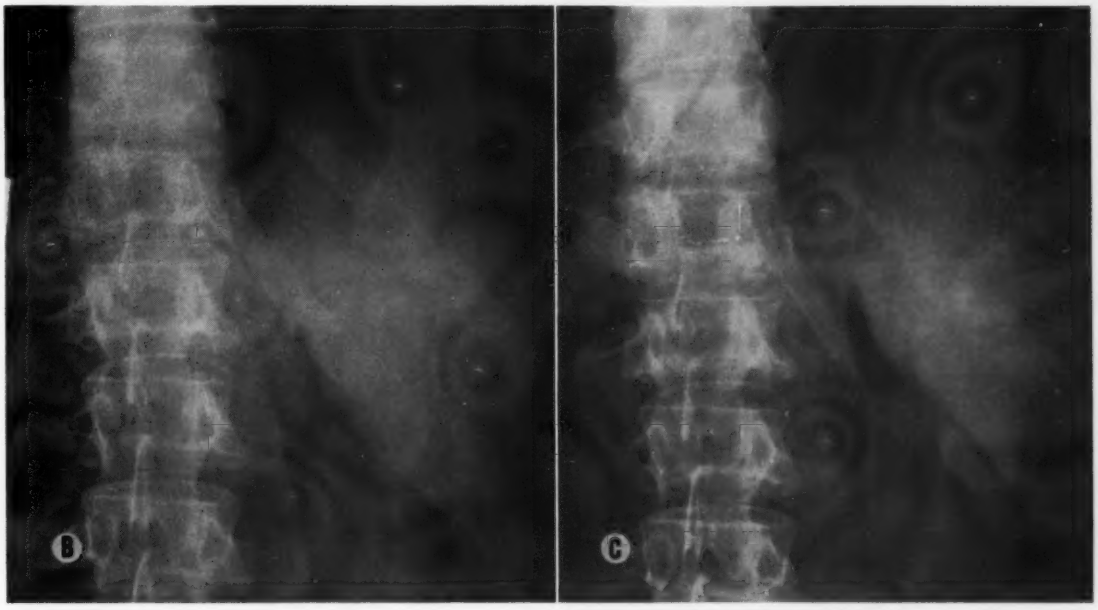

Fig. 1. Case 1. (*B*) Cholecystogram after 3 gm. of telepaque. No identifiable gallbladder shadow (October 7, 1953). (*C*) Cholecystogram after 6 gm. of telepaque. Gallbladder now faintly visualized (October 8, 1953).

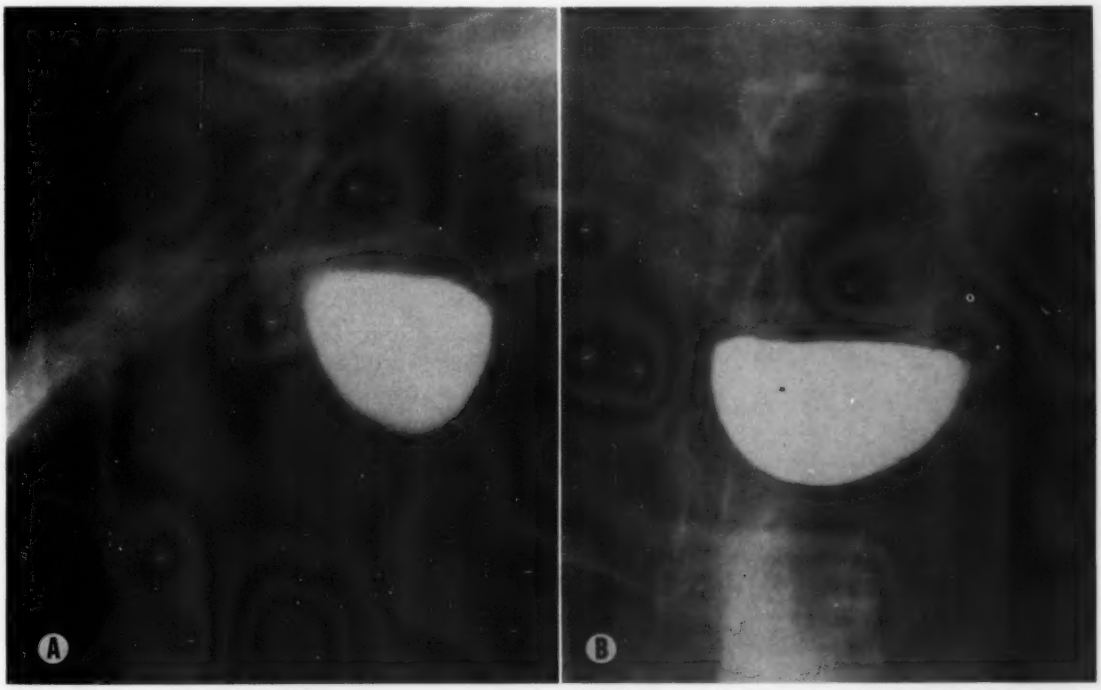

Fig. 2. Case 1. (*A*) Spot roentgenogram of thoracic inlet, lateral projection, demonstrating collection of telepaque in Zenker's diverticulum. No barium had been administered prior to this film (October 8, 1953). (*B*) Same as 2*A* in anteroposterior projection.

Fig. 3. Case II. (*A*) Preliminary roentgenogram of gallbladder area (November 23, 1953).

Massimiano[10] provide a comprehensive review, but the writer found no mention in the literature of the possibility that eosphageal lesions might be the cause of faulty opacification. Yet, in our group of patients there were two striking examples of "nonfunctioning" gallbladders produced by esophageal lesions encountered within a six weeks' period. These cases are presented here in detail.

REPORT OF CASES

Case I. T. D. M., a sixty-four year old male, was admitted to the Columbus Hospital for treatment of a protruding rectal mass. His only other complaint was frequency of urination. In the course of a routine work-up, he had a preliminary roentgenogram of the gallbladder area (Fig. 1*A*). Cholecystography, after 3 gm. of telepaque, resulted in nonvisualization (Fig. 1*B*). Preparation had been proper. As is the custom in this institution, a double dose examination was conducted on the following day and at this time there was faint visualization of the gallbladder (Fig. 1*C*). As soon as the cholecystography was completed, the patient was taken to the fluoroscopic room for examination of the stomach, and during preliminary fluoroscopy of the chest, a large, very dense opacity was observed in the midline of the upper mediastinum (Fig. 2, *A* and *B*). This was indeed startling and the fluoroscopist's first impression was that of ectopia of the gallbladder. The first

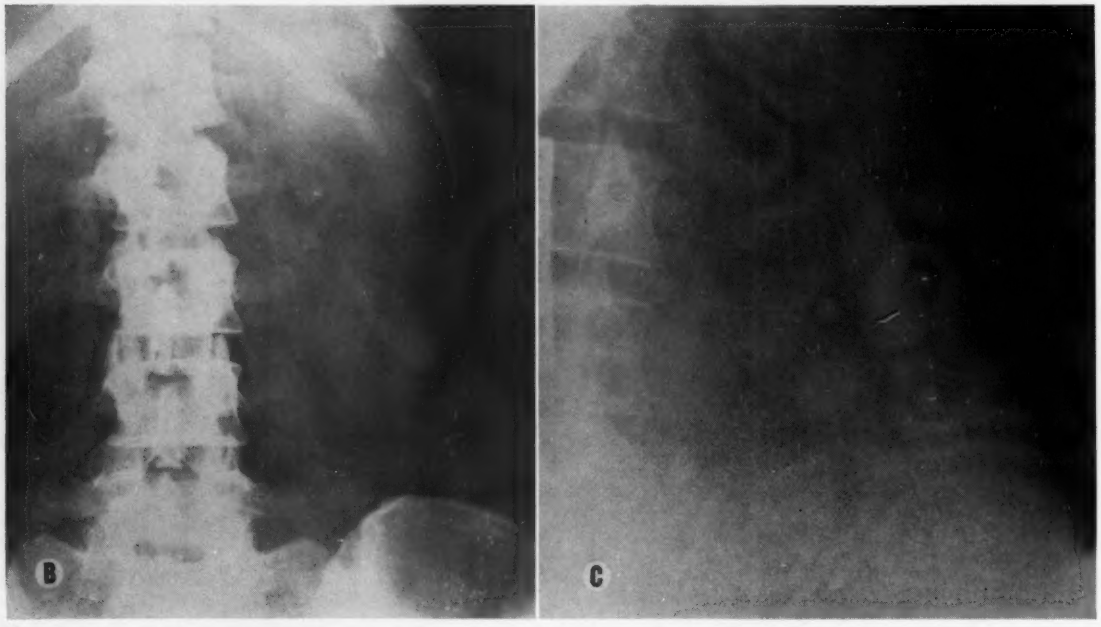

Fig. 3 Case II. (*B*) Cholecystogram after 3 gm. of telepaque showing poorly visualizing gallbladder. (*C*) Cholecystogram showing improved density of gallbladder after 6 gm. of telepaque (November 25, 1953).

swallow of barium disclosed a large Zenker's diverticulum in which the telepaque had collected.

Case II. M. L., a thirty-two year old female, a known diabetic and two months pregnant, was admitted to the hospital with the complaint of vomiting almost immediately after eating, the vomitus containing food eaten at a previous meal. She complained of no dysphagia. With proper diet and insulin, her diabetes was brought under control and she was discharged without roentgen studies, only to return four days later with the same complaints. At this time it was decided to investigate her gallbladder. The preliminary roentgenogram of the gallbladder area was made (Fig. 3*A*). After 3 gm. of telepaque, the gallbladder visualized faintly (Fig. 3*B*), and there was contrast material noted in the lower esophagus. A double dose examination was performed the following day which showed some improvement in the density of gallbladder (Fig. 3*C*), but again a considerable quantity of opaque material was present in an apparently dilated esophagus (Fig. 4). At this juncture, a barium study of the upper gastrointestinal tract was undertaken which confirmed the presence of a markedly dilated esophagus with the characteristic narrowing of the distal segment produced by cardiospasm (Fig. 5).

Fig. 4. Case II. Lateral roentgenogram of chest on November 25, 1953 after 6 gm. of telepaque showing considerable contrast material in lower esophagus.

Fig. 5. Case II. Lateral roentgenogram of chest after barium meal study demonstrating tremendous dilatation of esophagus with cardiospasm.

Not only do these 2 cases portray the fallacy of the assumption that failure to visualize represents failure of the gallbladder, but they also exemplify and justify the advice given by Robinson[11] as early in the era of oral cholecystography as 1931. He urged that the gastrointestinal tract be examined in conjunction with cholecystography in order that "faulty interpretation of shadows . . . may be reduced to a minimum . . . and in order that the faint shadow or absence of gallbladder shadow may have its proper significance."

SUMMARY

1. A consideration of the anatomic and physiologic mechanisms upon which cholecystography is dependent invalidates the categoric interpretation that the absent or faint shadow reflects a poorly functioning or nonfunctioning gallbladder.

2. The justifiable conclusions to be derived from faulty visualization are: (a) inadequate amounts of the contrast medium

have arrived at the gallbladder, or (b) the gallbladder has been unable to concentrate the opaque material presented to it, or (c) a combination of these factors is responsible.

3. To avoid misdirected medical or surgical treatment, the radiologist should confine his report to the observable facts: Poor visualization or nonvisualization.

4. Use of the terms "nonfunctioning" and "poorly functioning gallbladder" is misleading and should be abandoned.

5. Failure of the gallbladder to opacify warrants roentgen exploration of the gastrointestinal tract and careful questioning of the patient concerning proper execution of preparatory orders.

6. Two cases of esophageal lesions, a diverticulum and a cardiospasm, responsible for faulty visualization of the gallbladder are presented.

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THE BILIARY TRACT IN THE NORMAL AND CHOLECYSTECTOMIZED PATIENT

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IT IS now thirty-two years since the first cholecystographic examination was performed, and within the few years following the introduction of this new method certain criteria were established by which dysfunction or disease of the gallbladder was recognized. These criteria have stood the test of time and are fundamentally the same to-day as when first described. Various improvements in apparatus and roentgenographic technique have in the natural course of events improved the accuracy of the "Graham test" as it has come to be known. Now, however, due to the introduction of a new medium, biligrafin, it has become necessary to try and establish new criteria for a new type of examination—intravenous cholecystangiography.

The need for this is due to the fact that for the first time the biliary ducts are outlined by the contrast medium, together with the gallbladder. This is brought about by the high iodine content of the medium, which when excreted by the liver opacifies the ducts without the need for further concentration in the gallbladder which was necessary with the older media.

It has recently become obvious that the radiologist must be supplied with more information concerning, for example, the width of a normal common bile duct, as seen by this method and must be aware of the changes to expect in it when the gallbladder has been removed. In the past the ducts have usually only been visualized with the aid of surgery and the direct introduction of a cannula or catheter into them. When the ducts are visualized following operative interference, the accuracy of any measurements is open to doubt. Most of the measurements quoted in the literature concerning the size of the normal ducts are based on measurements obtained in this manner.

Consequently it was felt that a review of a series of intravenous cholecystangiograms might help to give some idea of the range of normality to be expected. With this in mind, I have deliberately avoided using any ancillary aid to improve visualization, such as the use of pethidine and a fatty meal prior to the examination as suggested by Aldridge.

This series is relatively small, and it is not suggested that the findings can be rigidly applied. When wider experience of the method is gained, it is hoped that it will be possible to state with more confidence what is normal and what is abnormal. However, I am of the opinion that the findings in this series show a fairly high degree of constancy under given circumstances and that it is possible to establish a working range of normality as a start to the firm establishment of these new criteria mentioned above.

The youngest patient on whom the examination was performed in this series was twenty-four years old and the oldest eighty-five years old.

One hundred and thirty-five examinations were performed on 125 patients, and of these an appreciable percentage were bedridden and quite a number of them were regarded as a bad risk from an operative point of view. The examination of these patients was in most cases satisfactory and demonstrates the need for little or no preparation of the patient prior to the examination.

The use of biligrafin has also one other advantage compared with the oral preparations in the elderly. Frequently, in this type of patient absorption of the oral media from the gastrointestinal tract is often incomplete and errors of diagnosis may be made due to nonvisualization of the gallbladder. By injecting biligrafin intrave-

nously one is sure that the medium has been delivered into the body in the prescribed quantity.

The technique used in examining these cases differed in detail from that recommended by the manufacturers (Schering A.G., Berlin). Forty cubic centimeters of the 20 per cent or 20 cc. of the 50 per cent solution were used routinely; films were exposed at fifteen minute intervals after injection up to one hour, then a further film was exposed at two and one-half hours. Following this a fatty meal was given to the patient and a further film exposed thirty minutes later to show the contraction of the gallbladder. Additional films were exposed where necessary after viewing the routine films while they were still wet. All films were exposed at a standard distance of 36 inches. In those cases in which a Graham's test had been performed and the gallbladder had not been visualized, a double dose technique using telepaque was tried before doing the cholecystangiogram.

In measuring the common bile duct, the maximum width reached by the duct during the course of the examination was recorded. In the case of the cholecystectomized patients, the maximum width of the duct below the junction of the left and right hepatic ducts was recorded. In these cases it was usually impossible to say where the hepatic duct ended and the common bile duct began.

The cases have been divided into two groups: *Group* I. All cases in this group have had no operative procedure performed on the gallbladder and *Group* II. All cases in this group have had a cholecystectomy at some time in the past.

The findings in the Group I cases have been set out briefly in Tables I–VI.

It will be seen from Table I that the average diameter of the common bile duct in the absence of disease was found to be 7.8 mm., or for practical purposes 8 mm. The range of normality appears to be from 7 to 9 mm. As mentioned above, this measurement is the maximum diameter seen on any film during the course of the examination, excluding the after fatty meal films. This range may appear at first to be small, but in fact it represents a 25 per cent variation from the normal average.

Measurement of the left and right main hepatic ducts where they were clearly seen shows that they are approximately half the diameter of the common bile duct.

The figures given for the diameter of the normal common bile duct are indirectly substantiated by the findings compiled in Tables II to V. It is very difficult clinically, due to many factors, *e.g.*, the pain thresh-

TABLE I

MEASUREMENTS OF THE COMMON BILE DUCT IN NORMAL CASES WITH AN INTACT BILIARY TRACT

Case No.	Diameter of Common Bile Duct in Mm.
1	8
2	8
3	8
4	8
5	8
6	8
7	7
8	7
9	7
10	9
11	7
12	8
13	8
14	7
15	7
16	9
17	7
18	9
19	8
20	8
73	8
74	7
75	9
76	7
106	8
107	8
108	8
109	7
110	8
111	8

Total number of cases—30.
Average diameter of common bile duct—7.8 mm.

Table II

SUMMARY OF FINDINGS IN CASES IN GROUP I IN WHICH GALLSTONES WERE PRESENT AND THE COMMON BILE DUCT EXCEEDED 9 MM. IN DIAMETER

Case No.	History of Jaundice	Pancreatitis
22	+	+
24	+	−
27	+	−
28	−	−
31	+	−
33	+	−
36	+	−
37	−	−
38	+	−
41	+	−
42	+	+
45	+	−
50	+	−
54	+	+
57	−	+
112	+	−
113	+	−

Total number of cases—17.
Total number having a history of jaundice—14.
Total number with pancreatitis—4.

Table III

SUMMARY OF FINDINGS IN CASES IN GROUP I IN WHICH GALLSTONES WERE PRESENT AND THE SIZE OF THE COMMON BILE DUCT DID NOT EXCEED 9 MM.

Case No.	History of Jaundice	Pancreatitis
21	−	−
25	−	−
34	−	−
35	−	+
39	−	−
40	−	−
43	−	−
51	−	−
52	−	−
59	−	−
61	−	−
62	−	−
114	+	−
115	−	−
116	−	−
117	−	−

Total number of cases—16.
Total number with a history of jaundice—1.
Total number with pancreatitis—1.

Table IV

SUMMARY OF FINDINGS IN CASES IN GROUP I IN WHICH THE GALLBLADDER WAS NOT VISUALIZED AND CONSEQUENTLY REGARDED AS PATHOLOGIC AND IN WHICH THE COMMON BILE DUCT EXCEEDED 9 MM.

Case No.	History of Jaundice	Pancreatitis
23	+	−
26	+	−
32	+	−
44	+	+
49	+	−
57	−	+
81	−	−
118	+	−
119	+	−

Total number of cases—9.
Total number with a history of jaundice—7.
Total number with pancreatitis—2.

old of the individual patient, and his or her accuracy as a historian, to decide with certainty whether a stone has passed down the common bile duct. It is also well known that a stone in the common bile duct does not necessarily produce jaundice. However, I think the findings in these tables are interesting.

If 9 mm. is regarded as the maximum diameter of a normal common bile duct, it is found that out of a total of 23 cases in Tables III and V there is only one case giv-

Table V

SUMMARY OF FINDINGS IN CASES IN GROUP I IN WHICH THE GALLBLADDER WAS NOT VISUALIZED AND CONSEQUENTLY REGARDED AS PATHOLOGIC AND IN WHICH THE COMMON BILE DUCT DID NOT EXCEED 9 MM.

Case No.	History of Jaundice	Pancreatitis
30	−	−
53	−	+
55	−	−
56	±	−
58	−	−
63	−	−
80	−	−

Total number of cases—7.
Total number with a history of jaundice—1 (doubtful).
Total number with pancreatitis—1

TABLE VI

COMPARISON OF FINDINGS USING TELEPAQUE AND BILIGRAFIN IN CASES IN WHICH TELEPAQUE HAS FAILED TO OUTLINE THE GALLBLADDER

Case No.	Biligrafin: gallbladder outlined + / gallbladder not outlined −
26	−
30	−
32	−
33	+
34	+
37	+
38	+
39	+
41	+
42	+
53	−
54	+
55	+
56	−
57	−
58	−
80	−
81	−
114	+
115	+
117	+

Total number of cases—21.

Total number in which the gallbladder was visualized with biligrafin but not with telepaque—12.

To date, 12 of these cases have come to operation, and the roentgenologic findings have been confirmed in all of them.

In addition to these cases, in 14 other cases the diagnosis was only possible with certainty by the improved visualization obtained with intravenous cholecystangiography.

ing a definite history of jaundice and one other with a doubtful history of jaundice, and only 2 cases with pancreatitis. On the other hand, out of a total number of 26 cases shown in Tables II and IV, in which the common bile duct exceeded 9 mm., it is found that 21 cases give a history of jaundice and 6 cases of pancreatitits. It is suggested from these findings that there is strong presumptive evidence of the passage of a stone down the common bile duct at some time in the past in the latter group of cases (Table II and IV) which has produced a dilatation of the duct. It would therefore seem that these groups of cases tend to confirm the maximum diameter of the common bile duct quoted previously as being 9 mm. in the normal intact biliary tract.

It was also observed in this group of cases (Group I) that in some of the patients in which telepaque failed to outline the gallbladder biligrafin succeeded (Table VI). In addition there were a number of cases in which the gallbladder was poorly outlined by telepaque, but with biligrafin visualization was improved to such a degree that a definite diagnosis could be made. The explanation for this lies in the fact that, provided biligrafin can enter the gallbladder, *i.e.*, provided the cystic duct is patent, the gallbladder will be visualized. Unlike telepaque, this medium does not depend on the concentration power of the gallbladder to produce a satisfactory shadow. However, if the gallbladder still retains a little of its function, a proportionately better shadow will be obtained compared with telepaque, due to the higher iodine content of the former.

Cholecystangiography was performed on 10 jaundiced cases in the hope that this

FIG. 1. Normal biliary tract.

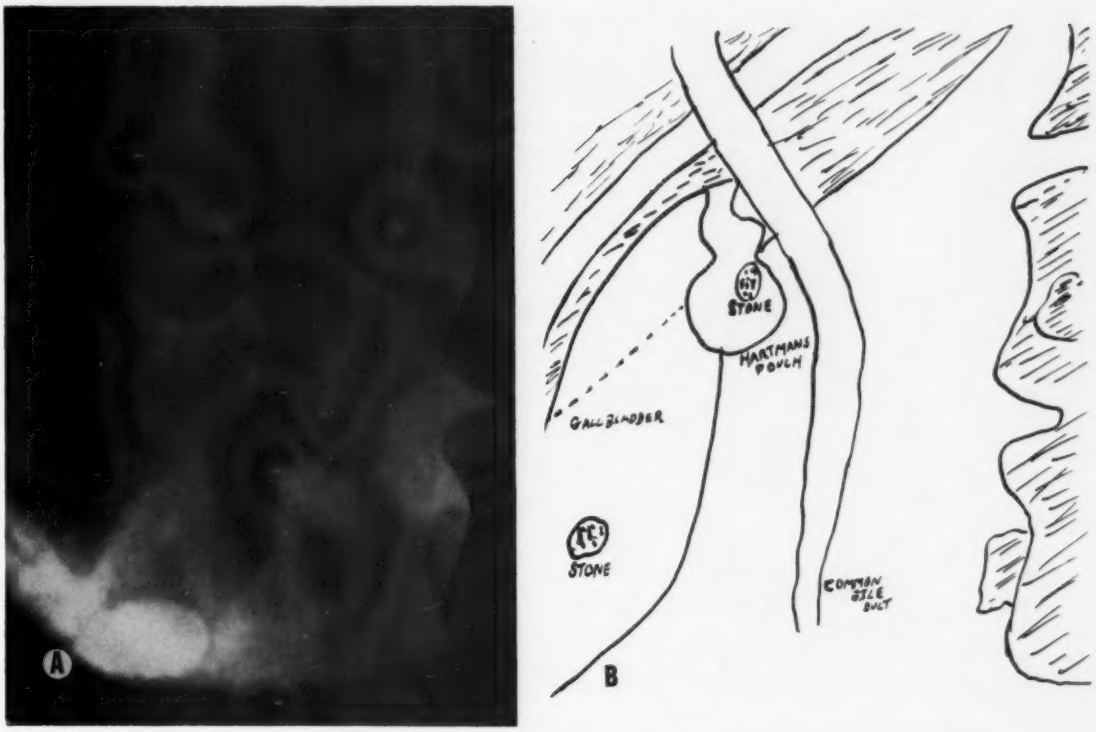

Fig. 2. Case 59. (*A* and *B*) This case demonstrates the formation of Hartmann's pouch.

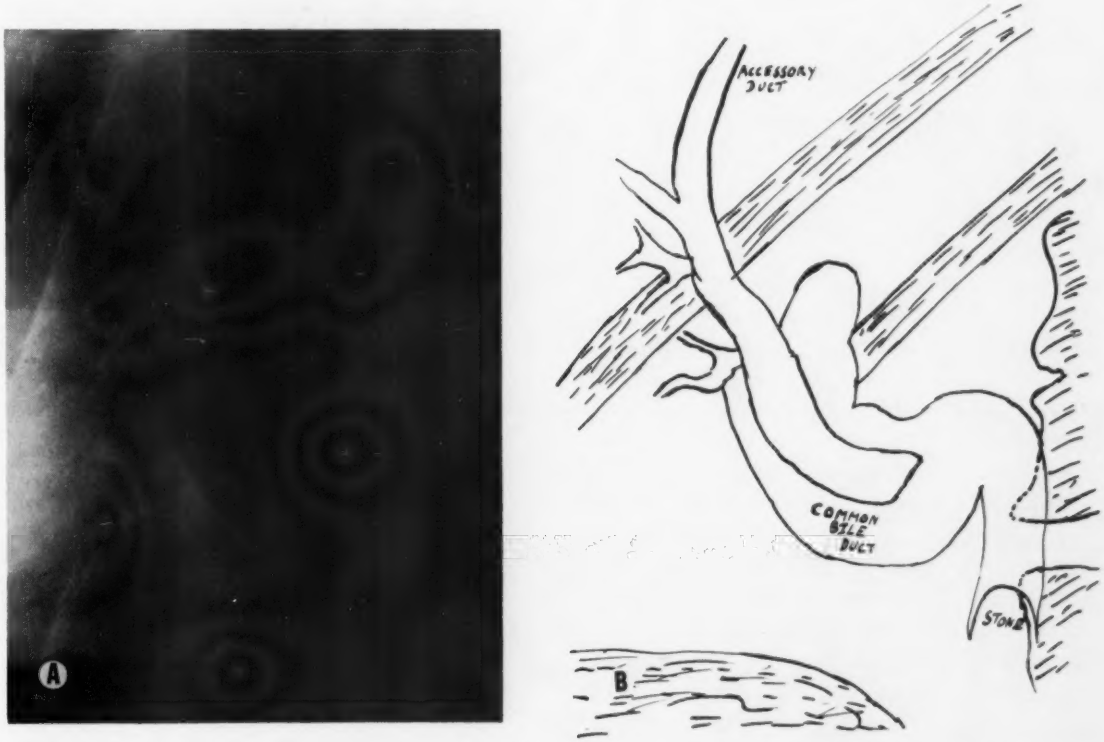

Fig. 3. Case 77. (*A* and *B*) The common bile duct is shown to be grossly dilated and there is a nonopaque stone in its lower part. An accessory right hepatic duct is also seen to be present. On later roentgenograms the gallbladder was visualized behind the common bile duct and was seen to contain nonopaque stones.

Fig. 4. Normal postcholecystectomy appearance of the biliary tract. This patient had her gallbladder removed thirty years prior to this examination.

new medium would be of some help. Although in some cases a faint outline of the bile ducts and/or the gallbladder was obtained, the contrast was never sufficiently good to produce films of a diagnostic quality.

From the results in the cases presented in Group I, it is possible to state with absolute certainty that, in the absence of severe liver damage, if the ducts are visualized but not the gallbladder then the gallbladder is pathologic and there is probably a stone blocking the cystic duct.

There are a number of individual cases in this group which are of particular interest. Case 59 demonstrates the formation of Hartmann's pouch. Case 77 illustrates a grossly dilated common bile duct, measuring 30 mm.; in addition an accessory right hepatic duct is also outlined by the contrast medium. The findings in both cases were confirmed at operation.

All the cases in Group II have had a cholecystectomy at some time in the past. Here it is far more difficult to establish normal values, due to the fact that such patients are rarely seen some time after operation unless they complain of symptoms or return with another disease. However, on reviewing the findings in 29 such cases, there seems to be sufficient evidence to cast doubt on the accuracy of some long held beliefs. It is suggested from a study of these cases that physiologic postcholecystectomy dilatation does not take place in the human subject. Reference to Table v shows that in 7 cases, of which 5 at operation were found to have blockage of the cystic duct with the formation of a mucocele, the common bile duct was not dilated. These cases can be regarded as cholecystectomized due to the blockage of the cystic duct, and this must have occurred at an appreciable time before operation for a mucocele to have formed. Fur-

Fig. 5. Cholecystectomized patient showing dilated common bile duct with a nonopaque stone in its lower part.

thermore, in 5 cases in which the operation was performed at varying periods from two to thirty years prior to this examination, the diameter of the common bile duct did not exceed 9 mm., *i.e.*, it was within normal limits for an intact biliary tract. It was also noticed that in those cases in which the common duct was dilated postoperatively without demonstrable pathology, there frequently appeared in the operation notes the statement that the duct was dilated at the time of operation.

In all the other cases in this group in which the common bile duct exceeded 9 mm. pathology was either found at operation or could be demonstrated roentgenologically, and in all these cases the patients complained of symptoms which could not be ascribed to any other system.

It would seem, therefore, that there exists sufficient evidence to doubt the theory advanced, particularly by Ivy, that the biliary ducts dilate following a cholecystectomy in the human subject. It is therefore suggested that any common bile duct exceeding 9 mm. must be regarded as abnormal, and that if no abnormality, *e.g.*, a stone, can be demonstrated in it roentgenologically, and such conditions as pancreatitis or a carcinoma of the head of the pancreas can be excluded, then the dilatation is due to the passage of a biliary calculus at some time in the past.

It is worth noting, both in the literature and in this series of cases, the high incidence of the postcholecystectomy syndrome and undoubtedly it is in this type of case that biligrafin will prove to be of the greatest value. Indeed, it is the only means for investigating such cases some time after operation.

CONCLUSIONS

1. A comparison of the value of the oral and intravenous methods of examining the biliary tract is discussed.

2. Intravenous biligrafin has been found to outline a significantly high percentage of gallbladders in which a double dose of telepaque has failed.

3. Intravenous biligrafin has been found to improve visualization of the gallbladder sufficiently well to make a diagnosis in a number of cases in which telepaque has only faintly outlined the viscus.

4. In jaundiced patients, although there is no contraindication to the use of biligrafin, the results are of little diagnostic value.

5. The average diameter of the common bile duct in a normal intact biliary tract as seen by intravenous cholecystangiography is 7.8 mm. The normal range appears to be 7–9 mm.

6. On reviewing the evidence presented above there would seem to be some grounds for doubting whether physiologic postcholecystectomy dilatation of the biliary ducts occurs in the human subject.

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My thanks are due to Dr. J. Winter and the staff of his department in which all these patients were examined and to Dr. P. H. Whitaker, to whom I am greatly indebted for the constructive criticism of this paper.

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INTRAGASTRIC GALLSTONE

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ENTRANCE of a gallstone into the intestinal tract by way of a cholecystoduodenal fistula is not unusual and approximately 1 to 2 per cent of all intestinal obstructions are due to this cause. However, passage of a gallstone into the stomach is extremely rare, and in a review of the available literature, we have been able to find only 2 cases of intragastric gallstone. In both of these cases, the stone passed from the gallbladder into the duodenum and then in a retrograde manner into the stomach. The purpose of this paper is to report a third case of intragastric gallstone, to our knowledge the first in which the gallstone entered the stomach through a cholecystogastric fistula.

REPORT OF A CASE

Los Angeles County Hospital, Case #270–349. A sixty-seven year old Caucasian male was referred to the hospital by his private physician on March 21, 1955. He gave a history of having had a bout of cramping epigastric pain and vomiting of "greenish material" one year previously. Several weeks before admission he had a similar episode which was accompanied by severe pain which radiated around the left costal margin to the back. This pain was so severe it caused the patient to "cry out." It recurred about every thirty minutes and lasted over a period of four days. Subsequently, his symptoms lessened but he continued to have mild, postprandial, mid-epigastric pains followed by hiccups. Both the pains and hiccups would subside following medication which, from the description, was probably tincture of belladonna. A roentgenologic examination of the upper gastrointestinal tract made a few days before admission was reported as showing a cholecystoduodenal fistula.

Physical Examination. The patient was a well developed, extremely obese, elderly Caucasian male who did not appear to be acutely ill. The blood pressure, pulse, respiration, and temperature were normal. A well healed, midline abdominal scar was present from a previous operation for a hernia. There was right subcostal tenderness with slight rigidity. There were no other significant physical findings.

Laboratory Examinations. The routine hemogram and urinalysis were normal.

Roentgen Examination (Fig. 1, 2 and 3). An examination of the upper gastrointestinal tract was performed on March 24, 1955, and repeated the following day. The esophagus was normal. At fluoroscopy, on both examinations, a well defined, somewhat lobulated, smooth mass was noted in the antrum of the stomach. This could be moved easily to the body of the stomach when the patient was in the supine position. The duodenal bulb filled well and appeared irregular near its apex suggesting the possibility of periduodenal inflammatory disease, but there was no evidence of an ulcer or fistula. A 1.5 cm. diverticulum was demonstrated at the junction of the descending and transverse portions of the duodenum. The roentgenographic impression was that of a bezoar or a pedunculated gastric polyp.

The gallbladder was not visualized by cholecystography. There were no radiopaque gallbladder calculi.

Gastroscopy. A large, dark green, sausage-shaped mass was seen lying in the greater curvature of the body of the stomach. It protruded about 2 cm. into the lumen of the

FIG. 1. Large filling defect in the antrum of the stomach.

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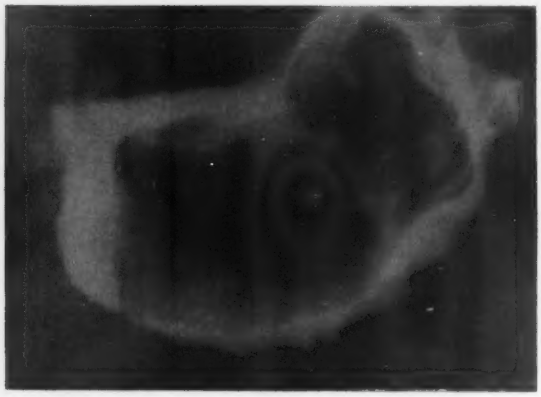

Fig. 2. Spot roentgenogram with pressure.

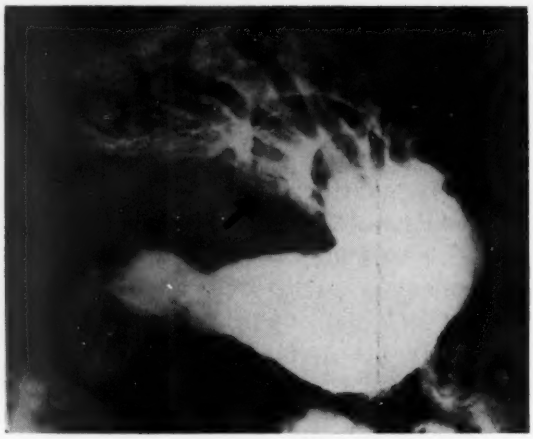

Fig. 3. Oblique projection showing the gallstone displaced into the body of the stomach (arrow).

stomach and was thought to represent a hemangioma.

Surgical Findings. A 9.0 by 6.0 cm. gallstone was found in the stomach. A cholecystogastric fistula, measuring 3.5 cm. in length and 1.0 cm. in diameter, was found communicating between the fundus of the gallbladder and anterior wall of the antrum. The gastric end of the fistula was closed. The gallbladder was thickened, encased in massive adhesions, and contained three large calculi. The common duct was normal and the cystic duct was extremely small.

DISCUSSION

The gallbladder may communicate with the duodenum, colon, jejunum, ileum and stomach by fistulae. Gallstones passing through these fistulae into the gastrointestinal tract lodge predominantly in the terminal ileum. The clinical course of gallstone ileus is well known and need not be discussed here.

The 2 previously reported cases of intragastric gallstones are similar in several aspects to the one presented here. Both patients were elderly males with long histories of gallbladder disease and in whom pericholecystitis associated with periduodenitis and cholecystoduodenal fistulae were found at operation. In these cases, the gallstones entered the stomach by retrograde passage from the duodenum. Our case is unique in that the gallstone entered the stomach through a cholecystogastric fistula. This event probably occurred at the time the patient experienced the long episode of severe recurrent epigastric pains which radiated around the left costal margin to the back. The fistula was not demonstrated on the upper gastrointestinal examination because the gastric end of the opening was closed.

It is well known that foreign bodies in the stomach can cause symptoms by irritation which leads to ulceration, bleeding, perforation and even obstruction. In our case the symptomatology was probably no different than one would have expected from cholelithiasis alone.

SUMMARY

A case of intragastric gallstone is recorded in which the gallstone entered the stomach through a cholecystogastric fistula.

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POSTOPERATIVE DIAPHRAGMATIC HERNIA: A COMPLICATION OF HIATUS HERNIA REPAIR*

REPORT ON TWO CASES

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AN UNUSUAL and serious complication which may follow surgical repair of a hiatus hernia is a dehiscence of the diaphragmatic counter incision with formation of a new hernia. It is the purpose of this paper to discuss the roentgenologic features of this condition and to present 2 recent cases in our experience.

Little has been written on this specific complication in radiologic or surgical literature. In a review of 111 consecutive cases of hiatus hernia repair Sweet[11] mentions one recurrence of hernia with a small peritoneal projection medial to the intact esophageal hiatus. Lam and Kenney[9] have not observed dehiscence of counter incisions in their presentation of 38 cases. Harrington[7] reports 1 recurrence of a congenital hernia in the diaphragm, and 9 recurrences of hiatus hernia but no dehiscence of counter incisions in a series of 430 cases. Allison[1] has had no dehiscence of the diaphragmatic counter incision in 48 operative cases. Boyd and Classen[2] do not record this complication in a review of 73 cases of surgical repair of diaphragmatic hernia reported in 1951. A more recent report by Boyd[3] points out the added hazard of making a diaphragmatic counter incision and a case in which dehiscence of the counter incision occurred twice is presented. However, Carter, Guiseffi and Felson[4] have reported 2 cases of postoperative diaphragmatic hernia following surgical division of the diaphragm in resection of the esophagus for tumor and for benign stricture, respectively. They also predicted that with more widespread employment of thoracic procedures involving the division of the diaphragm the potentialities for occurrence of postoperative diaphragmatic hernia would increase.

The commonest procedures involving division of the diaphragm are those associated with the esophagus, particularly hiatus hernioplasty and esophagectomy. One should be alert for this complication following these procedures.

This type of incisional hernia has been omitted in most classifications of diaphragmatic hernia. Most classifications[7,8] are divided into two large groups, the traumatic and nontraumatic types. Nontraumatic hernias include congenital and acquired varieties which occur at points of embryologic weakness. The traumatic hernias include those following direct injury, indirect injury or rupture following inflammatory necrosis. We agree with the suggestion by Carter, *et al*,[4] that an extension of this group should include those arising postoperatively from incision of the diaphragm.

Symptomatology of postoperative incisional diaphragmatic hernia will vary with the visceral relationships and their dynamic physiology. Symptoms in our cases consisted of pain, both abdominal and thoracic, dyspnea and tachypnea, nausea and vomiting, hematemesis, tachycardia, fever and shock. The symptoms may be very dramatic and suggest a chest condition rather than an abdominal one. Dysphagia and retrosternal fullness were absent. Both patients' symptoms were different from their preoperative hiatus hernia symptoms and were progressive. Both patients had a history of previous transthoracic hiatus hernia repair.

ROENTGEN FEATURES

The diagnosis should be suspected in all patients having had operative procedures

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involving the diaphragm who do not do well postoperatively.

Roentgenologic coverage should include both the chest and abdomen. Upright roentgenograms should be taken and the localization of air and fluid collections carefully determined. A double gas bubble or air containing loculus may be visualized indicating the supra and infradiaphragmatic portions of the stomach. This may have the appearance of a tilted hourglass with the constriction at the diaphragmatic defect. The gastric fluid level may be either in the infradiaphragmatic or supradiaphragmatic portion of the stomach, or a separate fluid level may be seen in each portion of the stomach. The supradiaphragmatic portion of stomach may be obscured by a pleural effusion.

Further studies should be made immediately in suspected cases by roentgenographic and fluoroscopic means using suitable radiopaque contrast media. A small amount of barium taken by mouth or tube may be enough to delineate the lesion and confirm the diagnosis. The nature and extent of visceral herniation through the diaphragmatic rent should be clearly evaluated. Adequate positioning and sufficient number of projections may show the location of the diaphragmatic defect and its relation to the involved viscus.

In following the progress of the ingested barium through the upper gastrointestinal tract, the location of the esophagogastric junction should be determined and differentiated from the incisional hernia. The possibility of a gastric volvulus and strangulation should be considered. If the colon is also involved, symptoms of partial or complete intestinal obstruction may appear in addition to those referable to the stomach. Barium enema study will help to establish the diagnosis and determine the amount of colon involvement.

The selection of a suitable medium for these contrast studies is not critical. A small amout of swallowed barium may be entirely satisfactory. The use, however, of water-soluble contrast media (such as miokon, urokon, etc.) may offer some advantage. These are clear, translucent solutions, which may permit the surgeon better visualization of the mucosa, if resection becomes necessary. Furthermore, they are more easily aspirated through Levine tubes than barium and do not lead to fecal impaction with dehydration in the colon.

In a convalescent patient single portable chest roentgenograms of poor technical quality may easily lead to erroneous conclusions and delayed diagnosis. We cannot overemphasize the need for adequate coverage with technically good roentgenograms.

Acute gastric dilatation of the supradiaphragmatic portion of the herniated stomach may achieve such massive proportions that the entire left hemithorax is occupied (massive gastric dilatation by itself may also do this). The thoracic stomach may be completely filled with either fluid or air. It may contain variable proportions of each with a demonstrable fluid level and may be confused with a hydropneumothorax. Mediastinal shift to the right is a frequent finding and may become marked.

DIFFERENTIAL DIAGNOSIS

The differential considerations in evaluating a chest roentgenogram showing a possible diaphragmatic hernia would include the following conditions: (1) hiatus hernia; (2) eventration of the diaphragm; (3) massive gastric dilatation with elevation of the diaphragm; (4) high diaphragm;[10] (5) left phrenic nerve injury; (6) hydropneumothorax; (7) pneumothorax; (8) empyema; (9) hemothorax; (10) pleural effusion; (11) atelectasis with elevated diaphragm; (12) tension cyst.

Of these, the differential diagnosis to be considered foremost in patients having undergone recent transthoracic hiatus herniorrhaphy, such as in the 2 cases presented here, would include a recurrence of the original hiatus hernia, hydropneumothorax (hemothorax, hydrothorax, pyothorax), massive gastric dilatation, and elevation of the diaphragm (with or without injury to the vagi or phrenic nerves), as well as a diaphragmatic hernia. Other possibilities

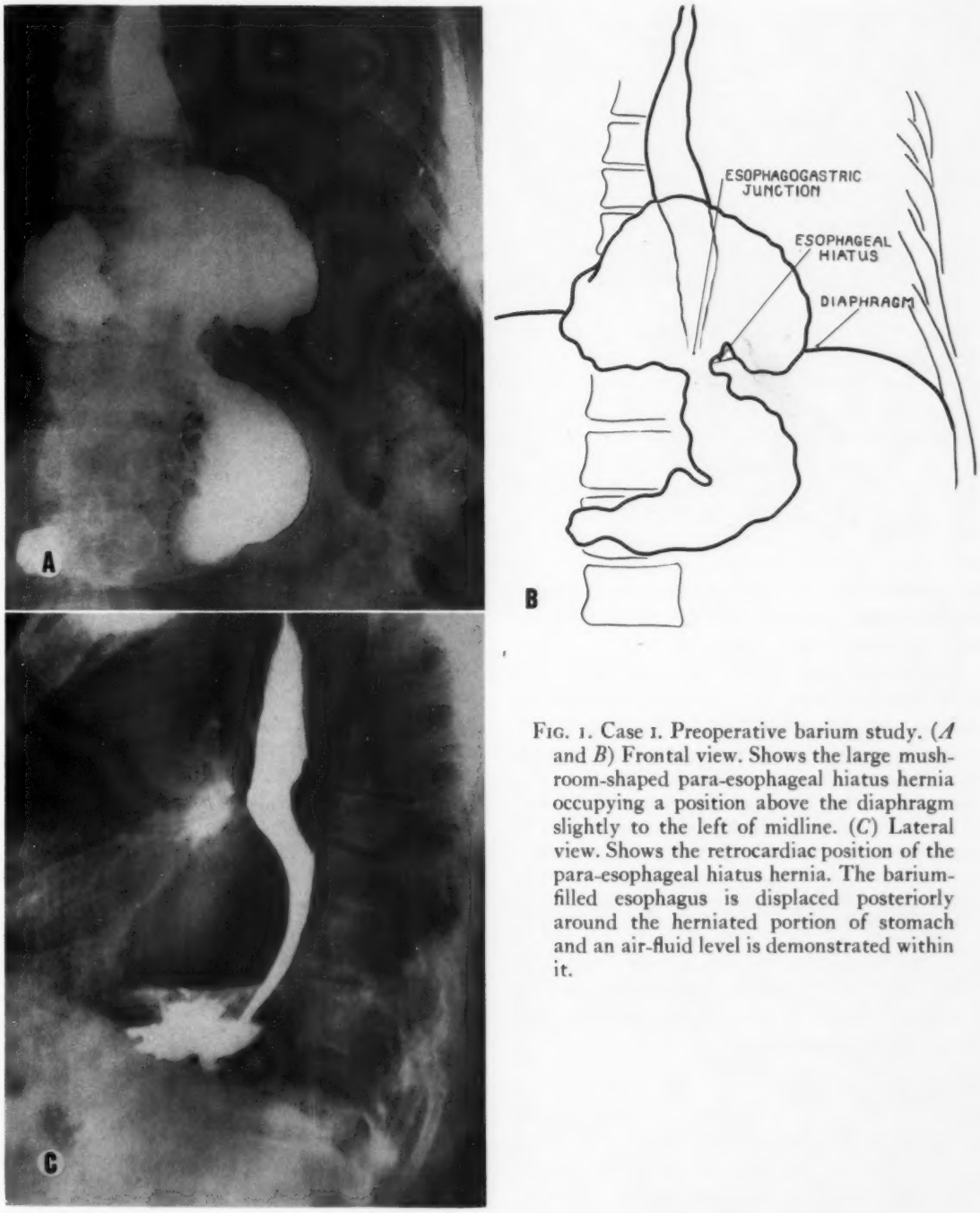


FIG. 1. Case 1. Preoperative barium study. (*A* and *B*) Frontal view. Shows the large mushroom-shaped para-esophageal hiatus hernia occupying a position above the diaphragm slightly to the left of midline. (*C*) Lateral view. Shows the retrocardiac position of the para-esophageal hiatus hernia. The barium-filled esophagus is displaced posteriorly around the herniated portion of stomach and an air-fluid level is demonstrated within it.

such as eventration of the diaphragm and tension cyst would have been precluded both in the preoperative evaluation and thoracotomy.

REPORT OF CASES

CASE 1. NYH No. 735688. This fifty-six year old white married male entered The New York Hospital for the first time with the chief complaint of inability to hold his food. Three years prior to admission a gastrointestinal series revealed a large para-esophageal hiatus hernia (Fig. 1). This was asymptomatic and no treatment was advised. The patient remained asymptomatic until four months prior to admission when he first noted difficulty in swallowing. At first this was minimal, but there was

a steady progression of dysphagia. Associated with the dysphagia was a substernal fullness and pressure. Immediately after eating the patient would become extremely nauseated. Induced vomiting would relieve the nausea and pressure. The patient changed his diet to soft foods and then to liquids. There had never been severe pain—either thoracic or abdominal. The vomitus had always consisted of recently ingested food. The symptoms were not relieved despite a milk and cream diet and medications.

Two weeks prior to admission another gastrointestinal series was performed and following this surgery was advised.

Physical Examination. The patient was a well-developed, well-hydrated white male of stated age in no acute distress. The temperature was 36.9°C., pulse 88, respiration rate 18, and blood pressure 130/80. Physical examination was not remarkable. The lung fields were clear to percussion and ausculation. The heart was not enlarged, rate 88, normal sinus rhythm, A_2 greater than P_2, no murmurs were heard. The abdomen was soft and nontender. No organs, nor masses were palpated.

Laboratory Data. Urinalysis was normal. The red blood cell count was 4,900,000, hemoglobin, 14.8 gm. and the white blood cell count, 9,700.

Course. Following admission and preoperative studies, including esophagoscopy, a transthoracic hiatus hernioplasty was performed under general endotracheal anesthesia. The counter incision in the diaphragm was closed with interrupted sutures of 00 black silk. The immediate postoperative condition was good. Levine tube suction of the stomach was maintained throughout the first postoperative day. The intrapleural catheter and the Levine tube were removed on the second postoperative day. The patient was ambulated and small amounts of fluid by mouth were tolerated well. A portable chest roentgenogram at this time was interpreted as gastric dilatation elevating the left hemidiaphragm and causing a mediastinal shift to the right. Despite the roentgen findings, there was no abdominal distention nor discomfort. The patient had moderate incisional pain on his third postoperative day and there was dullness to percussion and decreased breath sounds at the left posterior inferior lung field. This dullness continued on the fourth postoperative day and a thoracentesis was performed. Three hundred cubic centimeters of air and 150 cc. of serosanguineous fluid were removed. Some of the aspirate had the characteristics of gastric fluid. The Levine tube was replaced and gastric suction instituted following the thoracentesis. A slight temperature elevation to 38.2°C. occurred that evening.

On the morning of the fifth postoperative day the course changed. The patient complained of great weakness. The pulse was faint and rapid; apical cardiac rate, 180 per minute; blood pressure unobtainable; temperature elevated; and respirations shallow and rapid. Supportive measures were given and included Trendelenburg position, oxygen, intravenous fluids including whole blood (the hemoglobin had fallen 3.5 gm. since the first postoperative day), and a rapid acting digitalis preparation. A large caliber Levine tube was passed into the dilated portion of the stomach and 2,000 cc. of sanguineous gastric content removed. The patient's general condition improved slightly and bronchoscopy with suction aided the respiratory ability. However, the pulse, temperature, and respiratory rate remained elevated and the blood pressure continued below preoperative levels.

Supportive therapy was continued during the next two days. Digitalization was completed, with little change in heart rate. Temperature ranged from 38.5 to 40°C. and respirations continued to be rapid. Repeat bronchoscopy temporarily aided respiration. Several portable chest roentgenograms continued to demonstrate the large elevated stomach, mediastinal shift to the right, and increasing atelectasis of the left lower lung. The patient's condition appeared to be deteriorating, and on the eighth postoperative day a second thoracotomy was performed through the previous incision. The left hemithorax contained a large portion of the stomach that had herniated through a dehiscence in the diaphragmatic counter incision. A volvulus with clockwise rotation had occured in the herniated portion of the stomach, and the body of the stomach was gangrenous. The esophageal hiatus repair was intact. A sleeve resection of the gangrenous portion of the stomach was performed. The left upper abdomen was drained, the herniated stomach reduced, and the counter incision imbricated with 00 black silk mattress sutures. The patient appeared to be improved postoperatively for three hours, when he suddenly became disoriented and expired. Postmortem examination revealed a large embolus occluding the pulmonary artery. The esophageal hiatus, the diaphragmatic counter incision and the gastric anastomosis were all intact.

Postoperative Roentgenologic Studies. A post-

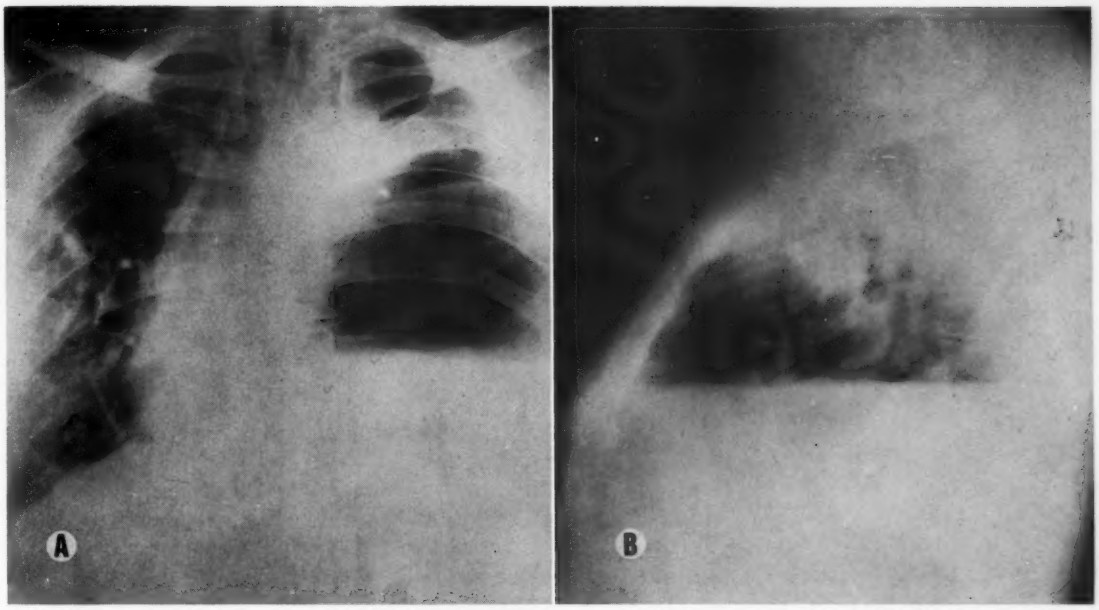


Fig. 2. Case 1. Portable chest roentgenogram on second postoperative day. (*A*) Frontal view. Massive distention of the herniated portion of stomach containing fluid and air and displacing the mediastinum to the right. (*B*) Lateral view. The herniated stomach occupies almost the entire left hemithorax.

operative chest film was obtained on the second postoperative day (Fig. 2) by means of portable technique. This showed a curvilinear dome-shaped density in the left hemithorax with a large air collection and fluid level below, suggesting a markedly elevated left diaphragm and massive gastric dilatation. The mediastinum was displaced to the right. Subsequent portable chest films showed slight increase in the degree of gastric dilatation and accumulation of fluid, and on the fifth postoperative day (Fig. 3) the entire left chest exhibited a homogeneous density.

On the sixth postoperative day (Fig. 4) a portable chest roentgenogram revealed the presence of a Levine tube which had been passed earlier. The tube passed through the esophagus into the stomach and continued its course laterally and upward into the left mid-chest with the tip of the tube at the level of the second anterior interspace. Two thousand cubic centimeters of gastric fluid had been removed through this tube and an air-fluid level was once again seen. These findings, it was felt, were consistent with a marked elevation of the left diaphragm and gastric distention; however, contrast studies were advised to evaluate this further. A water-soluble contrast agent, miokon, was then introduced through the gastric tube. Following this the esophagogastric junction was located adjacent to the tenth dorsal vertebra. The fundus and body of the stomach were identified within the left hemithorax and their appearance suggested a gastric volvulus.

Comment. In this case serial portable chest roentgenograms were taken. The initial findings showed a markedly dilated stomach apparently distended with large amounts of fluid and air and associated

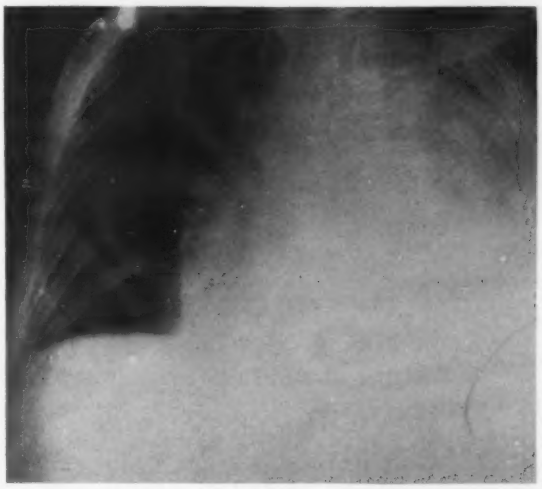


Fig. 3. Case 1. Portable chest roentgenogram on fifth postoperative day. Frontal view. Homogeneous density of the entire left chest, with the exception of the apex, due to tremendous distention of the herniated stomach and accumulation of fluid.

Fig. 4. Case 1. Portable chest roentgenogram on sixth postoperative day. Shows Levine tube in stomach passing laterally and upwards into the herniated portion. An air-fluid level has been re-established following fluid removal.

with marked elevation of the left diaphragm and mediastinal shift to the right. The dome-shaped margin superiorly was interpreted as diaphragm. Subsequent roentgenograms showed an increase in fluid accumulation within the stomach eventually causing the entire left chest, with the exception of the left apex, to appear dense. At this point Levine tube drainage was instituted, once again restoring an air-fluid level within the distended stomach. When next the miokon was introduced through the Levine tube, its appearance in the stomach suggested a gastric volvulus. The esophagus was of normal length and the esophagogastric junction was located in its normal position.

What had happened was that following the dehiscence of the large diaphragmatic counter incision anterolaterally, the larger part of the stomach had herniated into the chest, twisted slightly, and distended to fill the entire lower left chest, causing compression of the lung parenchyma and mediastinal displacement to the right.

Case II. NYH No. 602638. This fifty year old white married woman entered The New York Hospital for the fifth time complaining of epigastric pain and vomiting. One previous admission was for medical management of a hiatus hernia and the last admission was three months prior to the present admission for a transthoracic repair of a sliding hiatus hernia (Fig. 5). At operation a counter incision had been made in the diaphragm and this had been closed with interrupted sutures of 00 black silk.

Since operation the patient complained of postprandial epigastric pain. The pain was sharp and radiated to her back. The severity of the pain had increased ten days prior to admission. Persistent vomiting developed two days prior to admission. The vomitus was clear liquid until the morning of admission when emesis changed to coffee ground material. On the day of admission the patient visited the emergency room with the above complaints, and after roentgenograms were taken she was admitted.

Physical Examination. The patient was a well-developed, dehydrated, obese, white female acutely ill. The temperature was 37.5°F., pulse 80, respiration rate 22, and blood pressure 128/80. Physical examination revealed a healed recent left thoracotomy scar. The lungs were clear to percussion and auscultation and hypoactive bowel sounds were heard at the left posterior inferior lung field. The abdomen was soft, no tenderness, masses or organs were elicited.

Laboratory Data. Urine contained three plus acetone. The hemoglobin was 14 gm., hematocrit 43 per cent and the white blood cell count 9,800.

Course. After suitable hydration and preparation, a left thoracotomy was performed through the previous incision. Upon opening the chest one could see a large portion of the stomach, most of the body, except for the fundus and antrum, lying above the diaphragm. The counter incision made three months previously during the hiatus hernioplasty had dehisced and provided an opening through which the stomach had entered the chest. The esophageal hiatus repair was found to be intact and completely healed in its normal position medially, at some distance from the lateral diaphragmatic counter incision. The opening through the diaphragm measured approximately 4 inches in

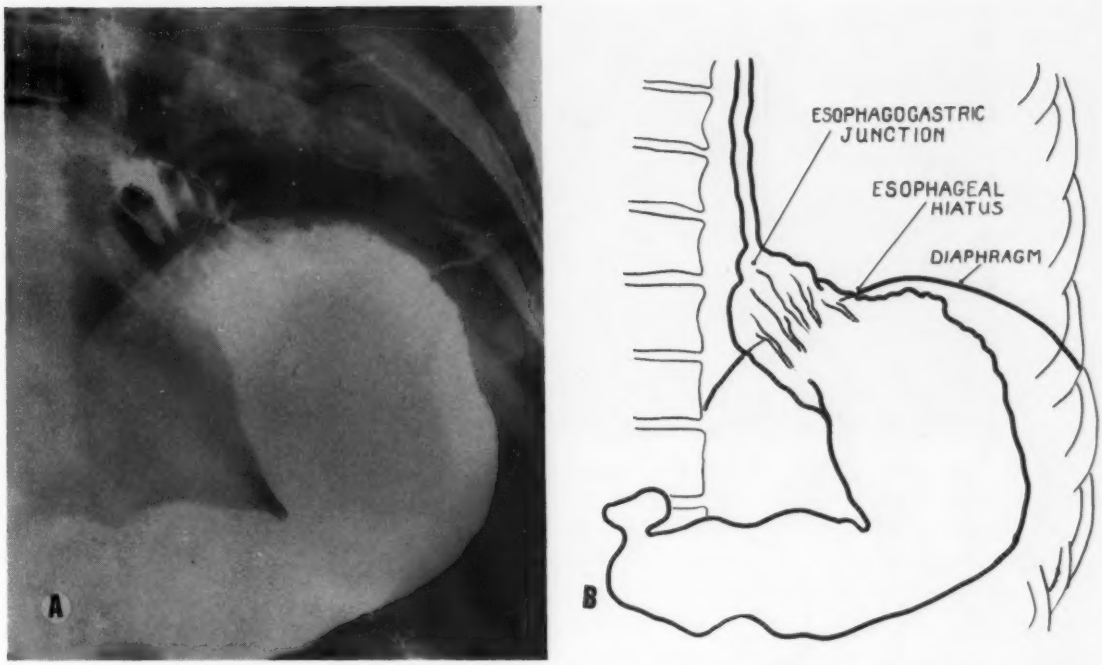


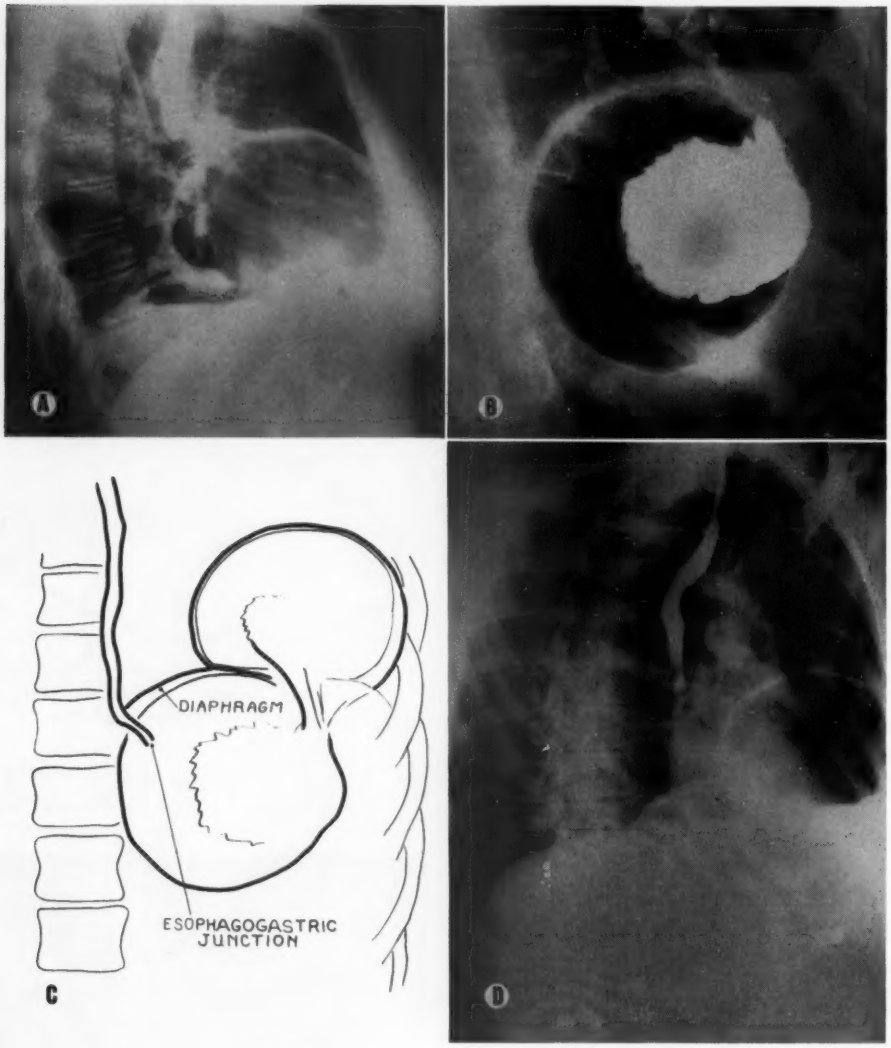
Fig. 5. Case 11. (*A* and *B*) Preoperative esophagram. Shows the large sliding-type hiatus hernia. Note the important relationships of esophagogastric junction and diaphragm.

length. The stomach was not strangulated although there was a slight volvulus. The color and appearance of the stomach was good, indicating that there had been no embarrassment of its blood supply. The hernia was reduced and the dehiscence of the diaphragmatic counter incision closed by imbricating with mattress sutures of co black silk.

The patient's post-operative condition was good and her hospital stay benign. She was discharged on her tenth postoperative day asymptomatic.

Roentgenologic Studies on Admission to Hospital (Three Months Post-Hiatus Hernioplasty). The patient was seen on the emergency service with the complaint of pain and vomiting. A posteroanterior chest roentgenogram was obtained which showed a large air loculus in the left lower hemithorax with a dome-shaped margin superiorly (Fig. 6). Further barium studies were then made with the patient in various upright and horizontal positions. These showed a normal esophagus which entered the diaphragm in normal position without evidence of a recurrent hiatus hernia (Fig. 7*A*). An air fluid level was present in the subdiaphragmatic portion of the stomach with clear delineation on multiple views of a herniated portion of stomach through the lateral leaf of the diaphragm into the left lower chest. Additional views with

Fig. 6. Case 11. Chest roentgenogram, three months post-hiatus hernioplasty. Frontal view. Shows a large air loculus at the left base.



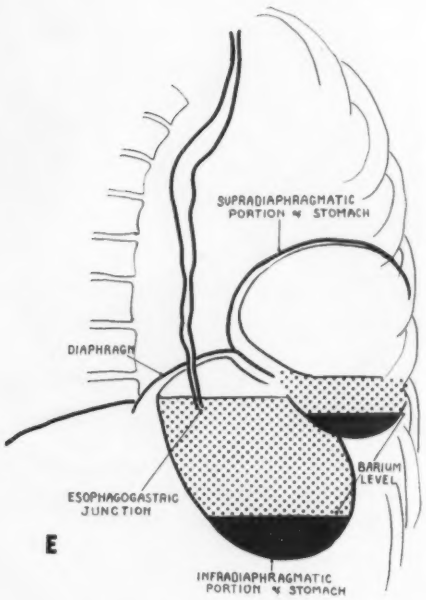


FIG. 7. Case II. Barium study following admission chest roentgenogram. (*A*) Lateral view following a barium swallow. Clearly demonstrates the defect in the diaphragm, constricting and producing the tilted hour-glass configuration of the herniated stomach. (*B*) Supine roentgenogram taken with the patient in moderate Trendelenburg position. Shows the barium pathway from the infradiaphragmatic portion into the supradiaphragmatic portion of the stomach. (*C*) Schematic drawing of relationships in *B*. (*D*) Oblique upright roentgenogram. Shows double air loculi with air-fluid-barium levels in each. (*E*) Schematic drawing of *D*.

the patient in the Trendelenburg position showed some of the barium to enter the supradiaphragmatic portion of the stomach (Fig. 7, *B* and *C*). Subsequent upright roentgenograms demonstrated the air-fluid-barium levels in both the supra and intradiaphragmatic portions of the stomach (Fig. 7, *D* and *E*).

In this case the diagnosis of diaphragmatic hernia was definitely established. Furthermore, the barium studies made it quite clear that this was not simply a recurrence of the original hiatus hernia, but rather a new herniation through the diaphragm anterolaterally at some distance from the esophageal hiatus.

DISCUSSION

The incidence of postoperative diaphragmatic hernia may increase,[4,6] with the increased frequency of thoracic surgery and thoraco-abdominal incisions. Dehiscence of the diaphragmatic closure, permitting herniation of abdominal viscera, is a serious complication endangering life. Early diagnosis and surgical repair is imperative. One death occurred in our 2 cases, as in the 2 cases of postoperative diaphragmatic hernia presented by Carter, Giuseffi and Felson.[4]

Obstruction and strangulation occur more frequently in traumatic diaphragmatic hernias,[5] and dehiscence of a small diaphragmatic counter incision can readily lead to such phenomena. Both occurred in Case I and only obstruction occurred in Case II. Obstruction of a stomach on Levine tube suction may be asymptomatic except for increased amounts of aspirate. Gastric dilatation secondary to obstruction may be confused with vagectomy effects after injury to the vagi in the hiatus hernia repair.

The prompt use of gastric drainage, either therapeutically or diagnostically, combined with careful roentgen examinations and bronchoscopy usually will reveal the diagnosis. Diagnostic needle thoracentesis is not advised within the first week to ten days postoperatively as adhesions have not developed between the stomach and diaphragm sufficiently to prevent leakage of gastric contents into the thorax.

The diaphragmatic counter incision must be carefully closed as its dehiscence is a more serious complication than recurrence of the hiatus hernia. Imbrication with mattress sutures of oo silk will probably result in a stronger repair than approximating the edges with interrupted sutures.

The earlier the complication occurs in the postoperative period the more difficult the diagnosis. The symptoms can be confused with the usual postoperative pain associated with thoracotomy. One hesitates to send a patient to fluoroscopy a few days following a major surgical procedure, but it is only with the closest cooperation of surgeon and radiologist that early diagnosis will be made. Both surgeon and radiologist must consider this complication in all patients having had incision of the diaphragm or the diagnosis may only be made at necropsy.

SUMMARY

1. Two cases of postoperative diaphragmatic hernia through a dehiscence in the diaphragmatic counter incision are presented.

2. This is a serious and progressive complication which can become an acute surgical emergency.

3. This complication should be considered in the differential evaluation of a patient following diaphragmatic surgery.

4. Imbrication with mattress sutures should be the method of choice in closing diaphragmatic incisions.

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Vol. 78, No. 4

GASTROINTESTINAL MANIFESTATIONS OF THE SCHÖNLEIN-HENOCH SYNDROME*

ROENTGENOLOGIC FINDINGS

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THE Schönlein-Henoch syndrome is characterized by a nontraumatic hemorrhagic diathesis with bleeding into the joints, skin, viscera, or any combination of these; and is associated with normally abundant platelets. The eponym is poorly chosen not only because purpura is a variable sign of the disease but because it was first recorded as a clinical entity by Willan[16] in 1808. His vivid description stressed the association of purpura, limb pain, and hemorrhages from various body orifices. Schönlein described peliosis rheumatica in 1837 as a combination of rheumatic joint symptoms and purpura; however, Henoch[11] is generally considered as the original observer of the co-existence of the dermatologic and gastrointestinal hemorrhagic manifestations. In 1914, Osler[14] described various forms of skin lesions seen in the disease and hypothesized an allergic etiology because of the condition's similarity to serum sickness. Whitmore and Peterson[15] were the first to describe the roentgenologic findings in the small bowel in 1946.

The etiology of the Schönlein-Henoch syndrome has been attributed to allergy. While various drugs, foods, and bacteria have been considered, an antigen has not been incriminated in all cases. Davis[5] reported that 10 of his 44 cases were preceded by tonsillitis. Antistreptolysin titers, however, in 8 of these 10 patients were negative. Gairdner[9] reviewed 140 cases in 1948 and found 71 per cent of these to be in males. Fifty-eight of these cases or 48.3 per cent were children under age of fourteen. Davis states that the sex incidence in children is equal. The youngest case of the modern literature is a two year old infant. Wintrobe[17] reports that the disease is never seen in newborns. While 3 of Willan's 17 cases were in infants, the exact ages were not stated, and it is possible that he was observing hemorrhagic disease of the newborn.

The principal clinical manifestations of this syndrome are purpuric exanthem, visceral hemorrhage and rheumatoid joint symptoms. Osler was one of the first to describe the 4 morphologic skin lesions as effusion, erythema, purpura, and necrosis. The rash usually consists of small, discrete, symmetric, urticarial eruptions over the extensor surfaces of the lower extremities and buttocks. Within hours, the lesions may become pink maculopapules which do not fade on pressure. Involvement of the abdomen, back, and upper extremities usually follows later.

Since the exanthem may or may not herald the disease, and since purpura may not be present at all, many authors have suggested dropping this term from the title of the disease. As we shall discuss later, the roentgenologic changes may precede the dermatologic manifestations.

Pain, secondary to involvement of the gastrointestinal tract including the appendix, may occur in any of the abdominal quadrants. Tenderness is often present but without muscular rigidity. Diarrhea or severe constipation may be found. Abdominal symptoms may appear prior to the purpura and mimic an acute abdomen. Fever is common but is usually moderate and rarely over 101° F.

According to Wintrobe[17] the hemorrhage is rarely severe enough to produce anemia. There is usually a polymorphonuclear leukocytosis, rarely an eosinophilia. Plate-

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lets, bleeding time, coagulation time and clot retraction are normal. The tourniquet test (Rumpel-Leede) may or may not be positive.

The entire gastrointestinal tract has been noted to be involved with the exception of the esophagus. Increased capillary fragility is responsible for extravasation of serosanguineous fluid into the intestinal wall with edema of the mucosa and submucosa. Extravasation into the mesentery and parietal peritoneum, associated with clear or blood stained fluid, has been reported. There is a relatively high incidence of intussusception. Brust[4] claims that the extravasation of blood into the intestinal wall causes local paresis and interruption of the peristaltic wave. This situation permits invagination of the uninvolved proximal segment into the paretic segment. Balf[2] describes the diseased bowel as having a scarlet, edematous appearance at surgery. Muldoon *et al.*[13] mention that the sharply demarcated involvement bears a resemblance to an infarcted loop. In these cases, however, the mesenteric vessels pulsate normally, eliminating the possibility of a major arterial obstruction.

Gairdner[9] observed endothelial proliferation of arterioles causing thrombosis. The primary disease is located in the smaller arteries and precapillary arterioles. The collagen is swollen and stains poorly. Fibrinoid degeneration is evidently the result of thrombosis and necrosis. All skin lesions, regardless of their age, manifest an acute inflammatory exudate about small blood vessels. These necrotizing arteriolar lesions provide a suggestive pathologic link with polyarteritis nodosa. In both conditions there may be crops of urticarial and purpuric skin lesions, edema, episodes of visceral symptoms and nephritis, and in both males are affected two to three times as often as females. Indeed, the Schönlein-Henoch syndrome and polyarteritis may be regarded as the same disease. The capillaries are involved in Schönlein-Henoch disease, the arteries in polyarteritis, and arteriolar lesions are common to both.

REPORT OF CASES

Case 1. I.L. (J.H.) No. 393671. A forty-two year old fur dealer was admitted with a seven day history of a skin eruption and epigastric pain of four days' duration. For a period of two months prior to admission, he had been taking various medicinals for weight reduction. These included amphetamine sulfate gr.$\overline{ss}$ t.i.d., atropine sulfate gr. 1/240 t.i.d., aloin p.r.n., and thiamine hydrochloride. He was allergic to many types of food, manifest by urticaria.

Several days prior to admission he developed a pruritic, spotty, papular eruption over both lower extremities. The rash gradually progressed over the next few days to include the buttocks, lower abdomen and arms. There was no response to benadryl. He felt well, in spite of the rash, until four days prior to admission when he developed a persistent epigastric ache radiating to the back and flanks. One day prior to admission, painful swelling of the ankles, right knee, and dorsum of the hands occurred. There was no history of fever, recent infection, hematuria, hematemesis, melena, neurologic manifestations or rheumatic fever.

Physical examination revealed a blood pressure of 140/90, pulse 90, respiration rate 18 per minute, temperature 101.4° F. There was a diffuse, purpuric, maculopapular eruption over

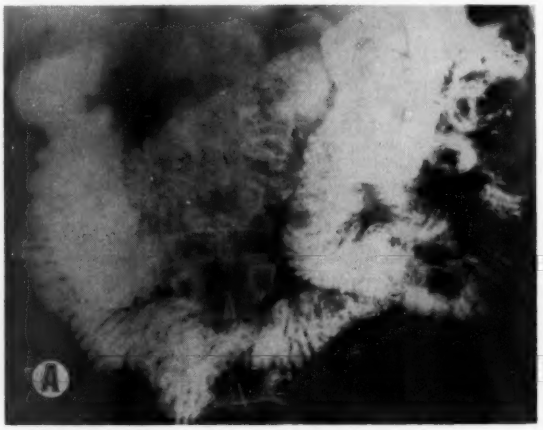

Fig. 1. Case 1. Upper gastrointestinal and small bowel examination performed on the ninth day of the disease. (*A*) The arrows in the region of the spinal column point to edematous mucous membrane of the third and fourth portions of the duodenum. Note the marked dilatation of the second portion of the duodenum. The arrows in the left upper quadrant point to an area of intussusception. Dilatation in all baroloid filled loops is evident.

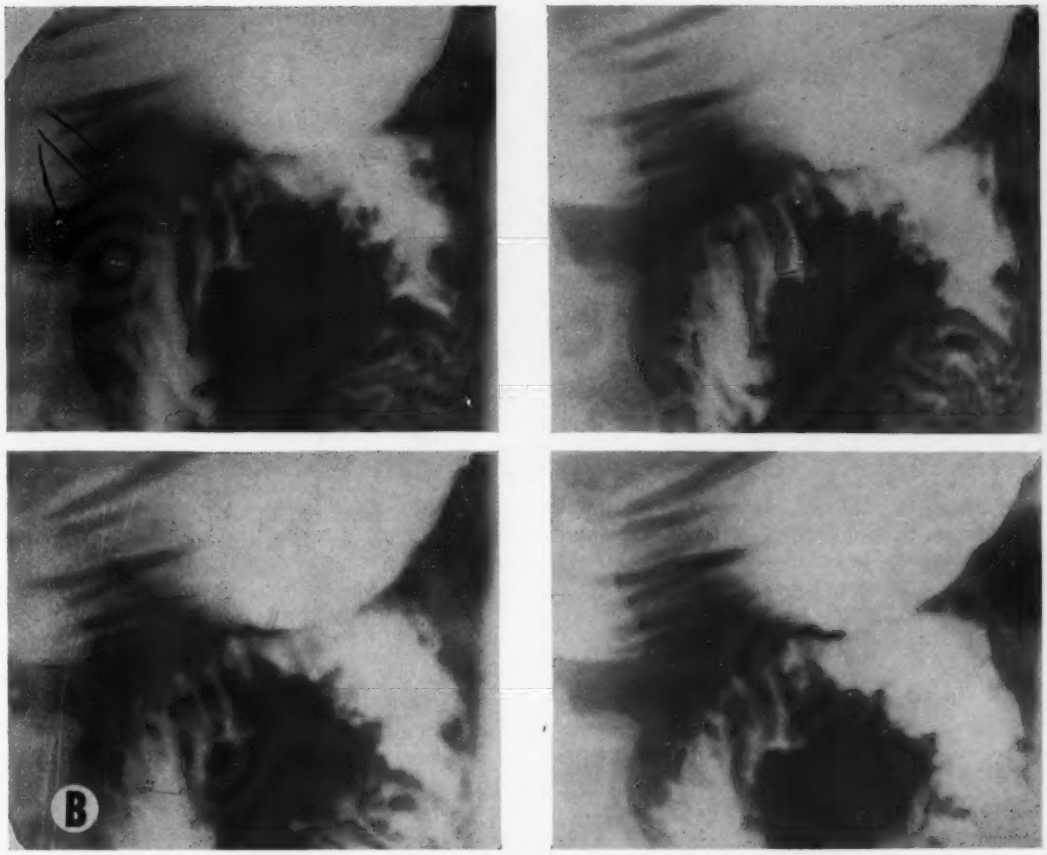

Fig. 1. Case 1 (*B*) Spot compression roentgenograms at the ligament of Treitz. The duodenal mucosa is edematous.

both upper and lower extremities. The primary lesion appeared to be a pin point sized papule. There was moderate tenderness over the epigastrium. No abdominal organ was palpably enlarged. There was edema over the dorsal aspect of the hands and feet. The right knee was greatly swollen, but there was no joint effusion.

The white blood cell count was 11,000 with a normal differential. No eosinophils were seen. The tourniquet test was positive; clot retraction and prothrombin times were normal. Bone marrow studies and platelet counts were also normal but these were done toward the termination of the disease. The antistreptolysin titer was reported as "a small amount present."

On the ninth day of the disease an upper gastrointestinal series and small bowel study revealed coarsened mucosal pattern in the second through the fourth portions of the dudenal sweep with an area of irritability just proximal to the ligament of Treitz (Fig. 1, *A* and *B*). The jejunal loops appeared dilated but the mucosa in this area was not remarkable. An area of transient intussusception was noted within the jejunum high up in the left upper quadrant. The head of the barium column reached an irritable cecum in three hours (Fig. 1*C*). At this time an air cast of the ileum and retention of barium in the jejunum were noted. The terminal 17 cm. of ileum was rigid, with marginal irregularities and showed a loss of normal mucosal pattern (Fig. 1, *D* and *E*). There was a tendency toward segmentation.

Diarrhea started on the tenth day and the patient was placed on 50 mg. of cortisone q.i.d. on the eleventh day. On the twelfth day his abdominal pain receded slightly but on the fourteenth day he began having mucoid, bloody stools. Proctoscopy on the fifteenth day revealed petechiosis of the sigmoid but no ulcerations. On the nineteenth day most of the purpuric lesions had cleared but some new purpuric areas were noted over the dorsum of both feet. He was maintained on cortisone and was asymp-

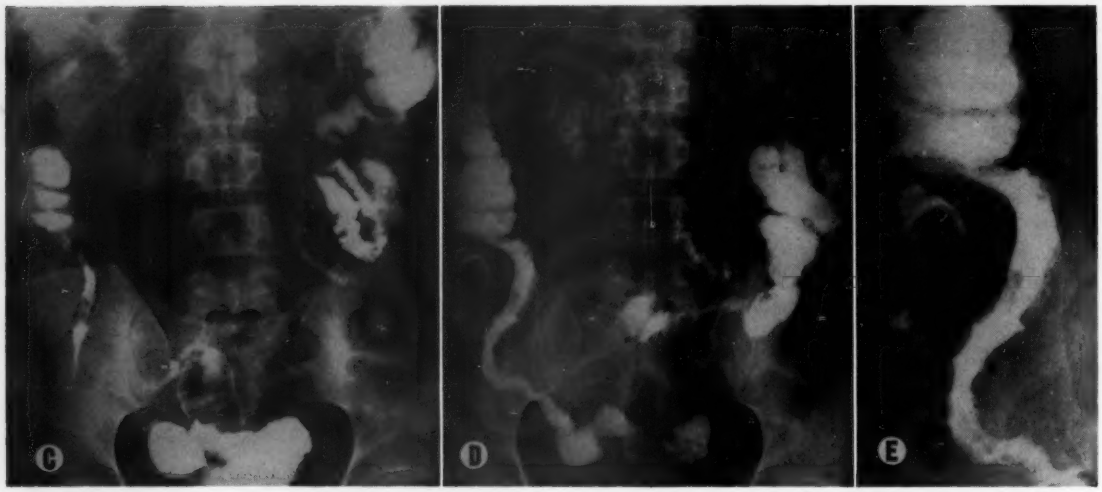

Fig. 1. Case 1. (*C*) The head of the barium column has reached the cecum in three hours. The terminal ileum is outlined by an air cast and is rigid in appearance. Note the dilatation of the ileal loops in the pelvis and retention of barium in the jejunum. (*D*) Three and one-half hour film. The cecum is contracted and irritable. Segmentation and narrowing of the mid ileum are present (arrows). Terminal ileal changes are better seen in Figure 1*E*. (*E*) Enlargement of Figure 1*D*. There is complete lack of mucosal pattern and marginal irregularity (arrows).

tomatic by the twenty-sixth day. The stool guaiac test remained positive. On the thirty-first day another exacerbation of purpura occurred but without pain. On the thirty-third day a repeat upper gastrointestinal and small bowel study again showed duodenal and jejunal dilatation (Fig. 2*A*) with a delay in transit through the jejunal loops. Another area of in-

Fig. 2. Case 1. Small bowel examination performed on the thirty-third day of the disease and two days following an acute exacerbation. (*A*) Jejunal dilatation, mucosal edema, and a transient intussusception (arrows) are shown. (*B*) Two and one-half hour film. The last several centimeters of terminal ileum appear normal, but proximally narrowing and edema are still apparent. Segmentation and flocculation are noted.

tussusception was noted. The location at this time was in a more proximal segment of the jejunum. On this occasion, however, the mucosal pattern in the jejunum was coarse. The appearance of the terminal ileum was changed (Fig. 2*B*). The terminal 10 cm. was no longer rigid and the mucosal pattern was now nearly normal in appearance. Segmentation and flocculation were more pronounced. On the thirty-fourth day all clinical signs of the condition had subsided.

Eight months after discharge, February 5, 1956, the gastrointestinal tract was again studied with the patient clinically asymptomatic. There was minimal exaggeration of the duodenal mucosa with duodenal and jejunal dilatation but less marked than on the preceding examinations (Fig. 3, *A* and *B*). The terminal ileum now appeared entirely normal but the transit time was essentially unchanged (Fig. 3*C*).

Case II. J.B. (J.H.) No. 385586. A forty-seven year old male was admitted on July 19, 1954. His history dated from June 22, 1954, when he was struck in the right inguinal region by a piece of metal thrown from a lathe. The swelling and discoloration that appeared immediately extended across the lower abdomen to the lateral aspect of the left thigh. Three daily injections of penicillin and streptomycin were given. On the third day of treatment, he developed crampy mid-abdominal pain with erythema of the lower extremities and inguinal region. Several days later these symptoms were followed by a generalized purpuric rash, vomiting, liquid black stools and asthenia. He had received penicillin once before, two years prior to admission, without allergic response. There was no previous history of allergy.

He was admitted to the hospital on the sixth day of illness with a clinical diagnosis of Schönlein-Henoch's purpura. The blood pressure was 150/100, pulse 80, respiration rate 20, and temperature 100° F. There were many purpuric lesions scattered over the body but principally below the waist. The ankles were edematous. No abdominal masses or tenderness were noted but there was slight resistance in the right upper quadrant. The Rumpel-Leede test was

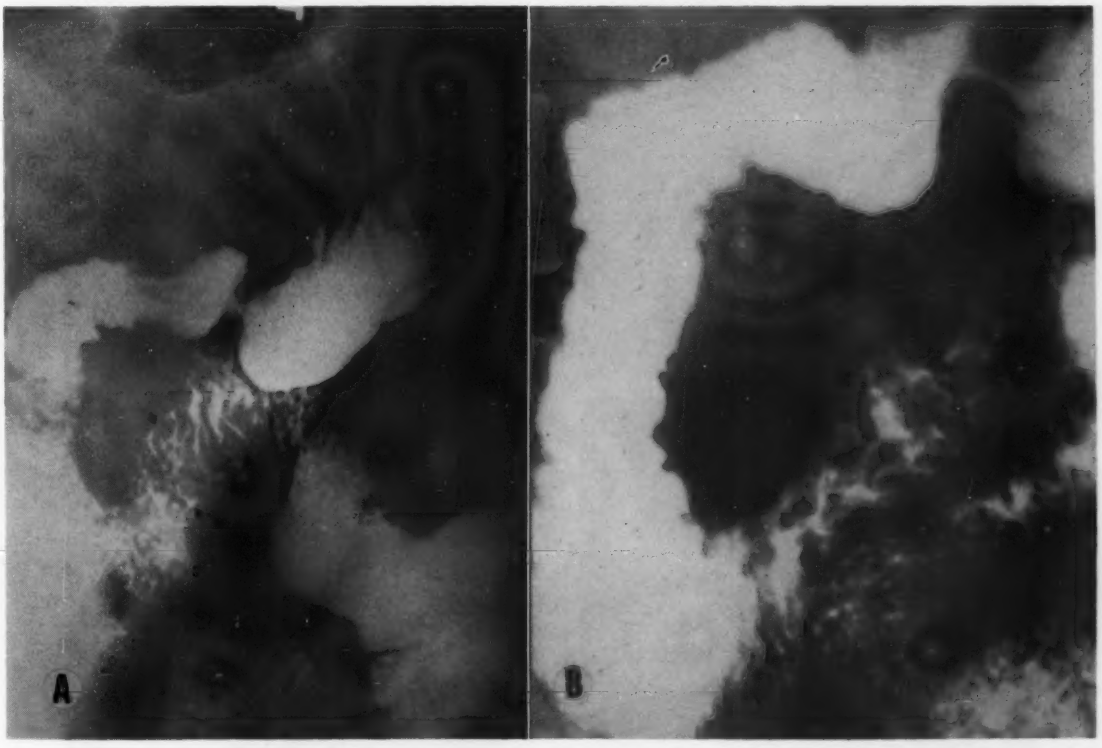

Fig. 3. Case I. Gastrointestinal and small bowel examination performed eight months after discharge when the patient was asymptomatic. (*A*) The duodenal mucosa is still minimally exaggerated and dilatation is evident in the duodenum and jejunum. (*B*) Enlargement of Figure 3*A*. Residual edema of duodenal mucosa is better shown.

positive (50 petechiae per 50 cm.^{2}). The stool showed a 4 plus guaiac reaction, red blood cell count was 4,870,000, hemoglobin 14 gm. per cent, white blood cell count 19,300, with 87 per cent polymorphonuclear neutrophils. No eosinophils were seen. The prothrombin time, bleeding time, and clotting time were all normal. A bone marrow aspirate taken on the eighth day of the disease was described as normal.

An upper gastrointestinal series was performed on the seventh day of the disease because of coffee ground vomitus. This study revealed marked narrowing, as well as obliteration and exaggeration of the mucosal pattern in the proximal jejunum (Fig. 4, *A* and *B*). The duodenum was also involved but to a lesser degree. Because of this jejunal abnormality and the fear of inducing additional bleeding, steroid hormones were not given. Three days after the initial gastrointestinal examination a small bowel examination was started but could not be completed due to the weakness of the patient. Spot compression roentgenograms of the proximal jejunum revealed return of mucosal pattern toward normal, but with marked residual edema (Fig. 5*A*). Generalized hypertonicity and prominent mucosal markings were still evident. Marked edema of a proximal ileal loop was noted (Fig. 5*B*). Sigmoidoscopy was negative on the thirteenth day. By the sixteenth day the Rumpel-Leede test was no longer positive. A repeat upper gastrointestinal series on the eighteenth day revealed residual coarsening of the mucosal pattern in the duodenum and jejunum (Fig. 6*A*) with delay of barium transit through these areas (Fig. 6*B*). The marked narrowing, however, was no longer visible. The transit time through the small bowel was now two hours. The patient was discharged on the twentieth day.

On June 11, 1955, ten months after discharge a comparative gastrointestinal study was performed. With the exception of apparent hypermotility, the examination was entirely normal (Fig. 7).

DISCUSSION

In Schönlein-Henoch's syndrome the

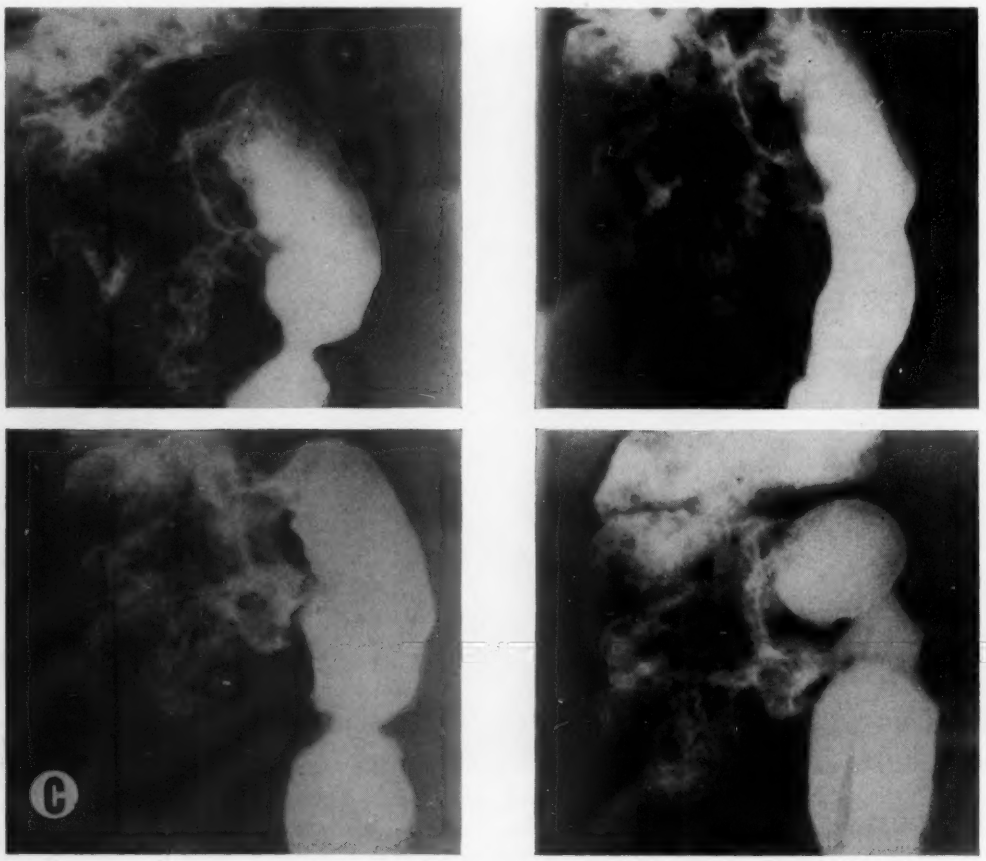

Fig. 3. Case 1. (*C*) Spot compression roentgenograms. The terminal ileum is now normal.

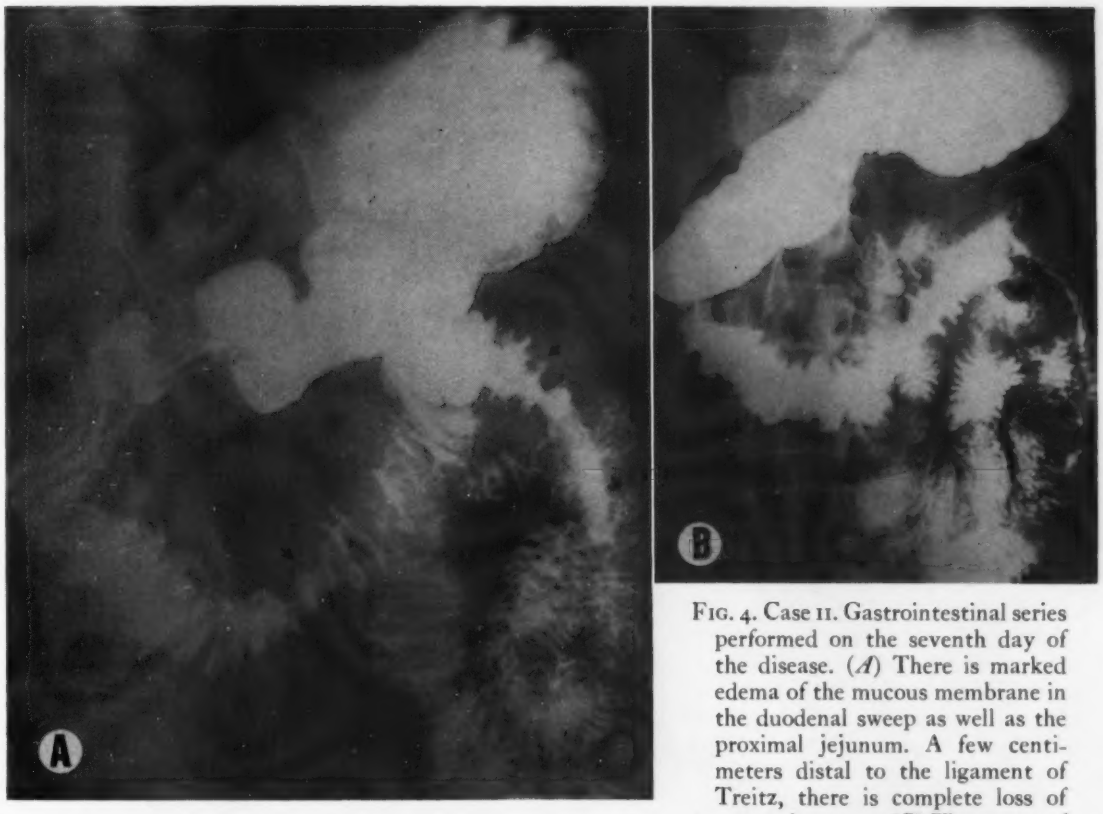

Fig. 4. Case II. Gastrointestinal series performed on the seventh day of the disease. (*A*) There is marked edema of the mucous membrane in the duodenal sweep as well as the proximal jejunum. A few centimeters distal to the ligament of Treitz, there is complete loss of mucosal pattern. (*B*) The extent of jejunal change is better shown.

entire gastrointestinal tract may be involved. Roentgen manifestations of the disease have been reported in the stomach, duodenum, jejunum and ileum. While there have been no roentgen changes reported in the large bowel, sigmoidoscopy revealed petechiosis of the sigmoid in Case I. Roentgenographic changes as previously described by Whitmore and Peterson,[15] Kraemer,[12] Berg,[3] Esposito,[6] and Fetter and Mills[7] resemble a disordered motor function pattern.[10] Our cases have been examined with baraloid, a colloidal barium preparation which is insensitive to small bowel content. Flocculation and other changes seen in disordered motor function, which may be due to small bowel content, rather than to afflictions of the neural pathway, would be less apparent than changes produced by mucosal edema and ulceration.[8]

Roentgenographic study reveals intestinal hypomotility characterized by delayed emptying of the stomach and prolonged transit time through the small bowel. Case I, however, had a small bowel examination during the acute illness and the transit time was normal. The gastric emptying time and transit through the jejunum were definitely delayed during the terminal asymptomatic phase in Case II, while transit through the entire small bowel appeared normal (two hours). Ten months later, the transit time was thirty minutes. Perhaps, then, the previous transit time of two hours was prolonged for this patient. Golden has hypothesized that an intact intramural nervous system is necessary for coordination of intestinal motion and transport of intestinal content. We have also seen changes in intestinal activity with inflammatory and neoplastic disease of the mesentery. The assumption, then, that serosanguineous effusion into these areas can be responsible for neural pathway interruption naturally follows. Other stigmata of the disordered motor function such

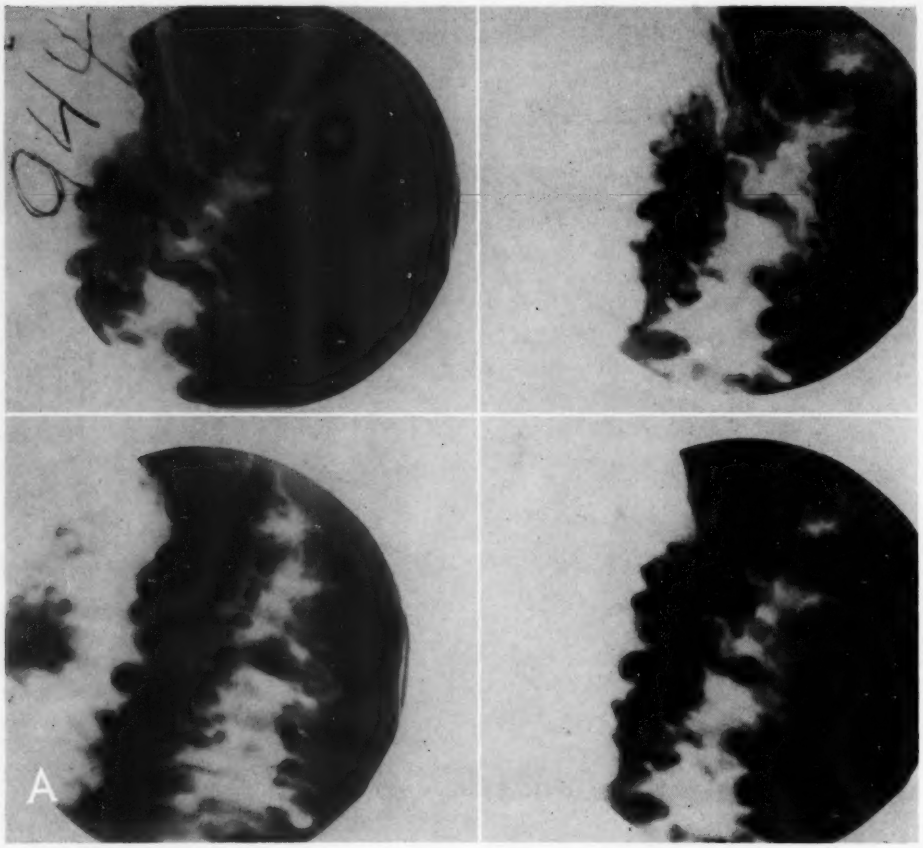

FIG. 5. Case II. Small bowel series performed on the tenth day of the disease. (*A*) Spot compression roentgenograms over the proximal portion of jejunum. There has been a striking return of mucosal pattern toward normal in the three day interval.

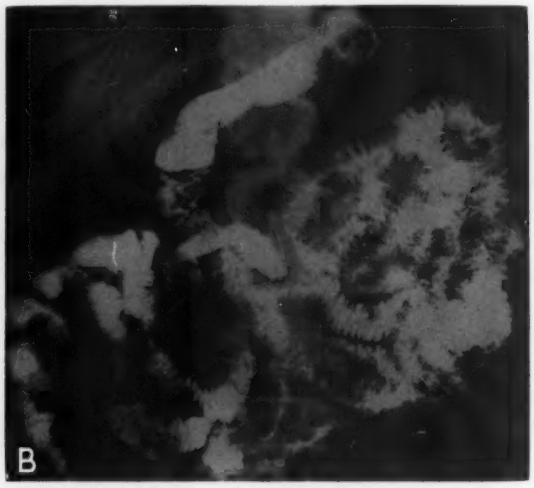

FIG. 5. Case II. (*B*) There is generalized hypertonicity and mucosal exaggeration. The arrows in the L5-S1 region point to marked edema and obliteration of ileal mucosal pattern.

as dilatation, hypertonicity, and segmentation were observed in our cases.

Submucosal infiltration of serosanguineous fluid has been postulated as responsible for coarsening and obliteration of the mucosal pattern. Marginal ulceration which may be due to obstruction of small end arteries folowing endothelial proliferation was suggested in the jejunum of Case II. Intussusceptions, perforations, and obstruction have been surgically described but to our knowledge not roentgenographically. Case I presented evidence suggestive of transient intussusception in the proximal jejunum.

The commonest site of involvement is thought by many to be the terminal ileum. Roentgenologically, although the number of examinations available for analysis is small, this does not seem to be the case.

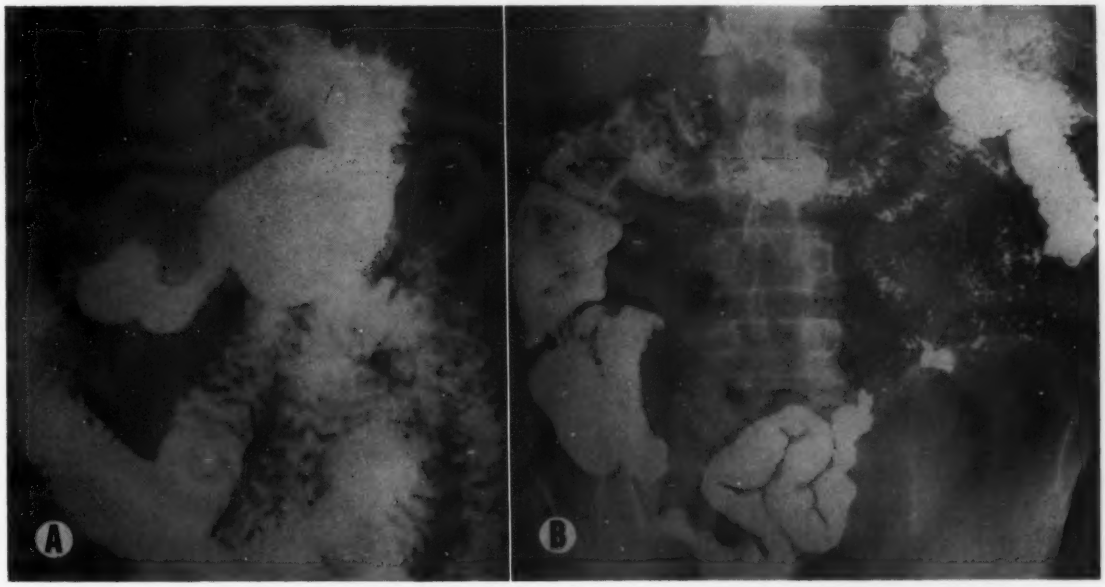

Fig. 6. Case II. Study performed on the nineteenth day. The patient had been asymptomatic for three days. (*A*) Minimal coarsening of the mucosal pattern is still present in the distal duodenum and proximal jejunum. (*B*) The transit time is two hours. Arrows point to residual barium in dilated jejunum.

The duodenum, jejunum, and ileum appear to be equally involved. Gairdner[9] has stated that he was impressed by the heavy male incidence among those cases which had developed some acute abdominal complications. With this in mind, it is extremely interesting to note that the 6 previous cases of Schönlein-Henoch syndrome with roentgenologic changes in the gastrointestinal tract reported in the literature as well as the 2 cases we are reporting are all males. It is also important to emphasize that all cases showing these changes in the small bowel manifested some degree of gastrointestinal bleeding. Althausen *et al.*[1] reported 5 cases with negative gastrointestinal roentgenographic examinations. Three were males and 2 were females. Not one of these 5 patients presented evidence of bleeding into the alimentary canal.

Fig. 7. Case II. Film exposed ten months after discharge when the patient was clinically well. The mucous membrane now appears normal (arrows). The transit time is thirty minutes.

The definitive diagnosis of Schönlein-Henoch syndrome cannot be made on the basis of the roentgen findings alone. A careful analysis of the clinical and laboratory findings is necessary. Diseases of the pancreas and biliary tract, primary intestinal disease and hypoproteinemia must be excluded. Hence, such laboratory data as serum albumin, electrolytes, stool for ova and parasites, stool fat, glucose tolerance curve and liver profile are extremely important factors to consider. The infantile

intestinal pattern should offer no particular difficulty since this physiologic finding is rarely present after the age of one year and this disease has never been reported under the age of two years with the exceptions noted. The transitory nature of the findings plus the clinical picture eliminate regional enteritis.

CONCLUSION

The clinical manifestations of Schönlein-Henoch syndrome are bizarre. Often the presenting symptom has been a nonspecific abdominal pain or diarrhea and the clinical diagnosis not contemplated until the onset of purpura. Whitmore and Peterson,[15] Kraemer,[12] and Esposito[6] have all emphasized the possibility of the presence of roentgenographic changes in the intestinal tract prior to the appearance of the dermatologic component. Althausen *et al.*[1] have stressed the fact that these cases may falsely present as acute surgical abdomens and Willan[16] as early as 1808 noted the nonfatal, self-limiting characteristics of the condition. The diagnosis, then of Schönlein-Henoch purpura should be strongly entertained when vague abdominal pain and gastrointestinal bleeding occur in a male with an allergic history whose small bowel shows primarily edema with a colloidal barium preparation.

SUMMARY

Two cases of Schönlein-Henoch purpura involving the small bowel have been presented. In each case, the striking roentgen feature has been the presence of areas of transitory edema. The causes of these changes have been discussed. The allergic nature of the condition and its pathologic similarity to polyarteritis nodosa has been stressed.

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Vol. 78, No. 4

ENTEROLITHS COMPLICATING REGIONAL ENTERITIS*

A REPORT OF TWO CASES

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THE formation of a primary calculus within the intestinal tract is an extremely rare occurrence. Most of the stones which are found in the intestinal tract are correctly assumed to have originated elsewhere, most probably in the gallbladder or biliary ducts. There are, however, well documented cases in which a stone discovered in the intestine has undoubtedly formed there. The principal condition which favors the formation of true enteroliths is stasis which is usually caused by diverticulum, inflammatory stricture or a stenosed segment of bowel due to adhesions or congenital atresia.

In the category of true enterolith formation are 2 cases, reported in this paper, where calculi developed as a complication of regional enteritis, and in 1 instance contributed to the development of intestinal obstruction. The association of enterolithiasis and regional enteritis has not previously been reported to our knowledge.

The roentgenographic findings are distinctive and have a bearing on the therapeutic management.

REPORT OF CASES

CASE 1. S. G., a thirty-six year old white male, was admitted to the Veterans Administration Hospital, Brooklyn, New York, for the second time in February, 1953, because of weakness. The patient's illness had begun twelve years earlier in 1941, when he developed diarrhea and lower abdominal pain. These symptoms persisted over the ensuing years. Barium studies of the small bowel revealed regional ileitis with involvement of the cecum. During the patient's first hospitalization in 1952, the presence of ileitis had been confirmed and impairment of kidney function was found. Excretory urograms showed a 2 by 3 cm. calculus low in the pelvis (Fig. 1).

On admission, physical examination revealed a markedly pale, fairly well-developed and well-nourished white male. The abdomen was slightly distended, with slight tenderness in the right upper and lower quadrants. There was a sausage-shaped mass palpable in the right lower quadrant, about 10 cm. long and 6 cm. wide. The spleen was palpable with the edge felt 3 cm. below the left costal margin. Bowel sounds were generally hyperactive. There was a left herniorrhaphy scar.

Laboratory Studies. The patient was anemic with a hemoglobin level of 9.7 gm. per 100 cc. Urinalysis showed small amounts of albumin, occasional granular casts and a specific gravity persistently below 1.012. The blood urea nitro-

FIG. 1. Case 1. Laminated calculus observed low in the pelvis. Excretory urogram, March 25, 1952.

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Fig. 2. Case 1. The same calculus observed to the right of the fourth lumbar vertebra. April 10, 1953.

gen was elevated to 38 mg. per cent, while phenolsulfonphthalein excretion was 13 per cent in one hour, indicating impairment of renal function. Guaiac tests for blood in the stool were positive up to four plus.

Repeated roentgenographic studies of the abdomen again disclosed the unusually mobile calculus. A gastrointestinal study showed no abnormality in the esophagus, stomach and duodenum. The characteristic roentgen findings of ileitis were present, manifested by alternate areas of narrowing and distention with irregularity of the margins of the bowel and with partial effacement of the mucosal markings in most of the involved segments. The cecum was deformed and contracted, and a large filling defect of its proximal portion was noted. The loops of bowel in the pelvis appeared to be matted together.

Intravenous pyelography on several occasions resulted in very poor visualization of the contrast material consistent with impaired renal function.

The patient's anemia was corrected by transfusions. On proctoscopy to a heighth of 20 cm. the mucosa appeared friable and bled easily. In early April, 1953, the patient began to have bouts of colicky abdominal pain and distention. An abdominal roentgenogram showed again a calcified body resembling a large kidney or gallstone. Its unusual mobility (Fig. 2 and 3) was somewhat confusing, and it was at first considered to represent a detached calcified epiploic appendage[4] or a calcified omental fat deposit.[22] However, during the course of a small intestinal study, a filling defect in one of the barium-filled loops of ileum was noted, and it was thought that the stone might be in the small intestine rather than in the peritoneal cavity (Fig. 4).

On April 14, 1953, the abdomen was explored under spinal and general anesthesia. Regional ileitis was found involving the terminal portion of ileum and the entire ascending colon. The area was markedly cicatrized and stenotic and was matted into a huge mass in the right pelvis. Resection of the terminal ileum and ascending colon with end-to-side ileotransverse colostomy was performed.

The pathologic specimen consisted of resected ileum (104 cm. in length), cecum, and

Fig. 3. Case 1. Roentgenogram demonstrating the extreme mobility of the calculus which is now in the left side of the abdomen. April 10, 1953.

ascending colon (16 cm. in length). The ileum showed segmental areas of thickening, ulceration, and hemorrhage. The ileocecal valve showed severe stenosis of its lumen and measured only 0.5 cm. in diameter. The terminal 15 cm. of ileum was extensively involved in an adherent inflammatory mass and was the site of a perforation which led into the mass. Great thickening of the mesenteric fat, together with a portion of the mass formed by the matted, diseased bowel loops, were responsible for the filling defects in the cecum.

A green, pea-sized nodule was found lodged at the ileocecal valve but did not obstruct the lumen. Approximately 40 cm. proximal to the ileocecal valve were two areas of stricture, measuring 0.5 cm. in diameter; the bowel in this region was intussuscepted and within the intussuscepted segment there was a black, bile stained foreign body measuring 2 by 1 cm. which could not be passed through the distal stricture. This foreign body represented the mobile calcified structure observed on the abdominal roentgenograms. It was oval in shape and extremely soft and friable. This, as well as the smaller body, on chemical analysis were composed of a mixture of calcium phosphate and urate. Postoperative abdominal roentgenograms showed no evidence of residual calculi in the abdomen. The smaller body found at the ileocecal valve undoubtedly represented a second enterolith containing insufficient calcium to make it visible roentgenographically. Microscopic examination of the bowel specimen showed ulceration with chronic inflammation consistent with regional enteritis.

Fig. 4. Case 1. The calculus is observed (arrow) in a diseased loop of distal small intestine which is partially filled with barium. April 11, 1953.

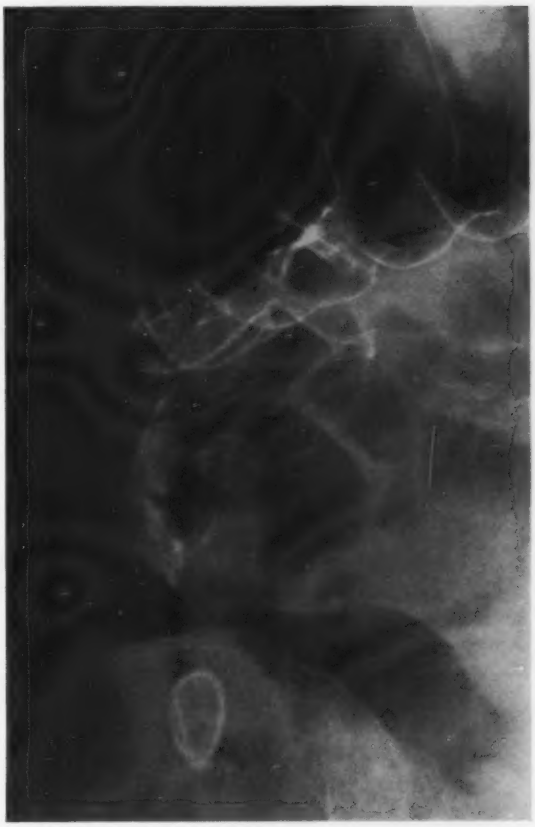

Fig. 5. Case 11. Air-contrast study of the colon with calculi widely separated in anastomosed loop of ileum (ileotransverse colostomy).

Case 11. J.W., a thirty-nine year old Negro male, was admitted to the Brooklyn Veterans Administration Hospital in November, 1951, because of abdominal pain. His illness dated back to 1943, when abdominal pain and distention led to two operations, a simple colostomy, followed several days later by an ileocolostomy.

In 1944, after another bout of abdominal pain and distention, a right hemicolectomy was performed. During the ensuing seven years the patient experienced intermittent abdominal cramps of a vague nature.

Two days before the present admission the patient noted the onset of abdominal cramps and constipation which persisted despite laxatives and enemas.

Fig. 6. Case 11. Calculi assume a varying position.

Physical examination showed a well-developed, well-nourished, thirty-nine year old Negro male, in no apparent distress. The abdomen was soft, nontender and not distended. No organs or masses were palpable. There were surgical scars in the right upper and right lower quadrants. Peristaltic sounds were normal. The remainder of the physical examination was not remarkable.

Laboratory findings were normal, except for the blood serologic examinations which showed a positive Kahn test as well as a Kolmer cardiolipin titer of 16 units. Spinal fluid studies and blood chemistries were normal.

Roentgenographic studies of the abdomen showed several small, rounded radiopaque bodies of calcific density in the right lower quadrant. These were extremely mobile, appearing in different positions in the abdomen in different films (Fig. 5, 6 and 7). There was no evidence of intestinal obstruction. A barium enema examination showed normal filling of the colon to the region of the hepatic flexure, but no retrograde flow proximal to this point.

A small bowel study revealed a normal appearing small intestine with an ileocolostomy in the region of the hepatic flexure. The previously noted calcific shadows were seen in the region of the anastomosis. Intravenous pyelograms and oral cholecystograms were normal, proving that the calcific shadows were outside the urinary and biliary tracts.

On February 12, 1952, the abdomen was explored under endotracheal cyclopropane anesthesia. The cecum and proximal ascending colon had previously been excised and the terminal ileum anastomosed to the distal ascending colon. There were many adhesions which produced kinking of the terminal ileum, and there

Fig. 7. Case 11. Excretory urogram showing the calculi approximated.

was marked stenosis at the anastomosis. There were two stones in the terminal ileum which could not pass into the colon because of the stricture at the anastomosis. The gallbladder was completely normal and contained no stones.

The terminal ileum and the residual segment of the right colon were excised and an end-to-end ileo-transverse colostomy was performed. The pathologic specimen consisted of a 30 cm. segment of ileum along with a segment of colon measuring 18 cm. At the junction of the small and large intestine, the bowel wall was thickened leaving a lumen only 0.4 cm. in diameter. Proximal to the anastomosis the mucosa of the ileum showed irregular hemorrhagic patches, while a perforation was noted 6 cm. from the proximal end of the ileum. Microscopic examination revealed acute and chronic inflammation of the ileum and colon. A foreign body granuloma characterized by fibrosis, giant cells, and infiltration with lymphocytes, plasma cells, and neutrophils, and considered typical of regional ileitis was found to involve the site of the ileocolic anastomosis.

The stones found in the ileum were subjected to chemical analysis and were found to contain a mixture of calcium phosphate and oxalate.

PATHOLOGY OF ILEITIS

Regional ileitis is a nonspecific inflammation of the distal loops of the small intestine, involving mainly the terminal segment and characterized by ulceration, proliferation, and cicatrizing granuloma formation. There is marked thickening of the intestinal wall and narrowing of the lumen. There is often segmental involvement with "skip areas" separated by areas of relatively normal intestine.[6,12,28] Complications include fistula formation and intestinal obstruction.[12]

Approximately 10 per cent of cases of regional ileitis go on to eventual partial intestinal obstruction of a subacute variety. The obstruction is the end result of many years of ulceration and scarring with gradual narrowing of the bowel lumen.[12] An acute exacerbation with edema and swelling can complete the closure and precipitate acute intestinal obstruction. In a similar manner, as demonstrated by the first of our 2 patients, a foreign body or enterolith may block the narrowed lumen and produce obstruction. Buckstein[7] describes a case of regional ileitis in which an ingested prune pit caused partial intestinal obstruction after lodging in the narrowed terminal ileum. This pit was visualized as a filling defect on barium study of the small bowel. Apparently it had not acquired a calcium layer which would have made it roentgenographically visible. Indeed enterolith formation occasionally takes place when a fruit pit serves as the nidus upon which chemicals are deposited.[20]

THE FORMATION OF ENTEROLITHS

Grettve[20] points out that chemical conditions favorable to the formation of enteroliths are always present because the intestinal contents have a high proportion of easily precipitable material. However, the normal alimentary tract is self-cleansing since peristalsis carries along and evacuates the material before significant concretions can form. Thus the essential cause of enterolithiasis is mechanical in nature. Physical changes, especially diverticula and bowel strictures, favor the retention of developing enteroliths. This, along with stagnation of the chyme, promotes the deposition of concrement-forming substances.[5]

The chemical composition of the enterolith depends on the acidity of the chyme at the site of formation. A high acidity, such as is found in the upper portion of the small intestine, enhances the precipitation of bile acids, especially choleic acid.[3,18,19,20] Concrements of this type are usually formed in diverticula, and, if large enough, can obstruct a segment of bowel where the lumen is of normal caliber.[18,32] It is noteworthy that choleic acid stones are radiolucent and therefore not visible on roentgenograms of the abdomen. Armitage, Fowweather and Johnstone[3] describe a case where a stone originating in a duodenal diverticulum was visible as a filling defect in the ileum after a barium meal.

An alkaline reaction is necessary for the precipitation of most calcium salts and this

is more likely to be present in the lower ileum. Calcium-containing stones are apt to be formed just above an incomplete obstruction,[20] as in Frink's case.[19]

Concretions found in the intestine may erroneously be regarded as gallstones, especially when they contain bile acids. Frink feels that "gallstone ileus," resulting from extrusion of a biliary tract stone into the intestine, is less common than we think and that many such cases actually represent true enterolithiasis secondary to diverticula.

Enteroliths may have as a core an ingested foreign body (especially a fruit pit) or undigested food particles, serving as a nidus for deposition of concrement. Enteroliths may be formed upon such nuclei as trichobezoars (hair balls), phytobezoars (food balls), trichophytobezoars (combined hair and food balls), and shellac bezoars.[23] Ingested medications, such as aluminum,[10] bismuth, or barium compounds[13] can form inspissated concrements containing calcium during passage through the alimentary tract. Nevertheless, Grettve states that enteroliths without a definite nucleus are more common than those with one.

SITE OF FORMATION OF ENTEROLITHS

While the absorption of water would actually seem to predispose to enterolith formation in the colon, there are only a few cases of large intestinal calculi on record. Grettve feels that this rarity may be explained by the fact that only those stones which are large enough to produce obstructive symptoms are discovered. Smaller ones are probably eliminated in the stool without being noticed. The ease with which stones may form in the colon is illustrated by Harland's case in which 166 calculi were found in a large intestine in which megacolon had developed proximal to a narrowed segment of the sigmoid.[21]

The content of the small intestine, in contrast to that of the colon, is liquid throughout. The material in the terminal ileum is of the same fluid consistency as that at the pylorus. Nevertheless, case reports of true enteroliths of the small bowel, though rare, are more frequent than those of the colon, although the likelihood of precipitation in the former is far less.[3,13,14,18,19,20,23,32] The relatively smaller lumen of the small intestine, as compared with that of the colon, may be a factor in the arrest of calculi and the increased frequency of their detection in the small bowel.

A Meckel's diverticulum is an example of a blind pocket where calculi may form. At least 12 such cases have been reported.[12]

Fecaliths of the appendix, which are the most numerous of all intestinal calculi are probably the best example of the influence of stasis in stone formation.

Enterolithiasis in the newborn has been reported on several occasions. The case reported by Khilnani and co-workers[25] was associated with anal atresia. Camp and Roberts' infant[8] had stenosis of the terminal ileum and Trump's patient[34] had two high jejunal stenoses. Intrauterine ingestion of fatty vernix caseosa and calcium in the amniotic fluid may predispose to calcium soap deposition[25] and concrement formation. Bowel strictures or atresia prevent normal transport of meconium and predispose to calculus formation.

ROENTGEN DIAGNOSIS

Inasmuch as enteroliths will have developed in patients with regional enteritis only after the disease has existed for a long period of time, it hardly seems likely that cases will be seen where the diagnosis of regional ileitis has not already been established. Thus, in a patient with regional ileitis the presence of a freely mobile abdominal calculus should immediately suggest that the calculus is within the small bowel and probably proximal to a point of inflammatory stricture. On the other hand, patients may occasionally be seen in whom the diagnosis of regional ileitis has not been made but in whom the disease process has existed sufficiently long for a calculus to have formed. Hence, it is necessary to review the differential diagnosis of freely mobile abdominal calcifications.

True enteroliths must be differentiated from the following:

1. Biliary calculi which have entered the gastrointestinal tract or which are contained in an unusually mobile gallbladder.
2. Appendiceal calculi.
3. Calcified omental fat deposits.
4. Free calcified intra-abdominal bodies including: (a) detached calcified epiploic appendages, (b) calculi which have been extruded into the peritoneal cavity from intestinal diverticula, the appendix or the gallbladder, and (c) calcified lipogranuloma of the peritoneum.[35]

ARCHITECTURE OF STONES

True enteroliths possess the characteristics of lamination, smooth borders and extreme mobility, but these qualities are neither distinctive nor diagnostic. They may resemble gallstones or appendiceal fecaliths and have also been mistaken for urinary calculi. Round or oval in shape, they possess a smooth, dense, heavily calcified outer shell and a relatively radiolucent center. They may be facetted. Roentgenographically the enterolith is indistinguishable from calcified omented fat deposits and calcified epiploic appendages.[4,22,29]

In Palmström's[30] case a calculus which had formed in a Meckel's diverticulum was somewhat distinctive in appearance. Although it had not been demonstrated preoperatively, a roentgenogram of the specimen revealed that it was oval-shaped with a constricted waist and two bulbous extremities. The smaller or proximal portion was thought to have formed in the diverticulum itself, while the larger distal portion was thought to represent a portion which had projected into the lumen of the ileum and which had grown larger due to accretion by deposition of chemical elements of the chyme. Finally, the entire calculus was extruded into the lumen and caused obstruction.[30] Thus, a calculus possessing such a contour may be suspected of having originated in a diverticulum. In general, however, calculi which are thought to have originated in a Meckel's diverticulum are facetted and resemble gallstones according to Allen and Donaldson, who surveyed all reported cases of calculi found in Meckel's diverticula.[1]

MOBILITY

Morales[29] states that it may actually be impossible to make a completely certain roentgen differentiation between those calculi which are contained in a very mobile organ, such as a segment of the intestinal tract and a free, intraperitoneal stone such as a detached, calcified epiploic appendage. This will probably prove to be the case in most instances unless the intraluminal calculus can be outlined by contrast methods, as occurred in our first patient, where the stone in the ileum was fortuitously demonstrated as a filling defect during a barium contrast study of the small intestine. Successive roentgenograms taken with the patient in different positions will show that the stone moves in relationship to fixed structures. This is due mainly to mobility of the mesentery, but there may also be longitudinal motion of a stone within the bowel lumen as a result of peristalsis and antiperistalsis.

Only a few other conditions show similarly mobile calcific shadows. Calcified omental fat deposits can show remarkable shifts in position, as shown in the cases reported by Holt and MacIntyre.[22] A calcified epiploic appendix may become detached and remain freely movable in the peritoneal cavity, as reported by Barden.[4] Morales[29] also presented a series of cases in which calcified bodies, representing detached appendices epiploicae, showed marked mobility within the abdomen.

DIFFERENTIATION FROM GALLSTONES

Gallstone ileus can mimic true enterolithiasis with obstruction.[15,27,31] Since the stone is in the bowel lumen, there is considerable mobility observed on serial films. However, there will usually be a history of gallbladder disease probably with previous roentgenologic demonstration of gallstones or gallbladder dysfunction. Furthermore,

at the time of intestinal obstruction roentgenograms of the abdomen may show air or barium in the biliary tract. Presumably a gallstone large enough to obstruct the bowel can reach the intestinal lumen only by erosion directly from the gallbladder or biliary ducts. The resulting fistula permits air or contrast medium to enter the biliary system.

Figiel *et al.*[17] pointed out that in 10 of 17 cases of gallstone ileus a correct diagnosis could be made on a preoperative roentgenogram of the abdomen. However, not all intestinal gallstones are immediately manifested by ileus or reliable symptoms of intestinal obstruction. Nor is air in the biliary tree always readily identified. In these cases a barium meal study to demonstrate an internal biliary fistula should be done.[17]

In 3 other cases reported by Figiel and his co-workers, a correct diagnosis was made after a barium meal examination. The three pathognomonic signs of gallstone obturation are: obstructive ileus, ectopically located biliary calculus and air or barium in the biliary system. In the absence of definite proof of cholelithiasis, it is hazardous to label as gallstones all concretions which are discovered in the intestinal lumen. Chemical analysis is not conclusive since both gallstones and enteroliths may contain calcium as well as bile acids and pigments in considerable quantity.

DIFFERENTIATION FROM APPENDICEAL CALCULI

The surgical and radiological literature contains numerous case reports of appendiceal calculi noted on preoperative roentgenographic studies.[11,16,23,26,33] There is almost invariably associated appendicitis, occasionally chronic but usually acute, with frequent perforation. Thus, the indentification of an appendiceal calculus is usually an indication for surgery. Statistically, a laminated calculus in the right lower quadrant will usually prove to be an appendiceal stone.[16]

Appendiceal calculi may also show mobility on serial roentgenograms.[11] Felson and Bernhard[16] demonstrated fluoroscopic mobility in 8 of 15 patients with roentgenographically visible appendiceal stones. These authors present an excellent discussion of the differential diagnosis of this condition. Lamination and mobility of appendiceal fecaliths can mimic true enteroliths but Felson and Bernhard state that the latter tend to be larger and less opaque and may be surrounded by a thin layer of gas. This finding was not evident in our cases.

SUMMARY

Two cases of regional enteritis in which enteroliths were discovered, in one of which the stone contributed to the development of intestinal obstruction, are described. The pathogenesis of enterolithiasis is discussed, especially in relation to stenosing lesions of the intestine and stasis in diverticula and the appendix. The roentgenographic finding of a mobile calcific density is highly suggestive of an enterolith, but other conditions producing mobile, intra-abdominal calcifications such as fecaliths and gallstones must be considered in the differential diagnosis.

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ILEOCECAL INTUSSUSCEPTION IN THE ADULT

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INTUSSUSCEPTION may be defined as the invagination or telescoping of one part of the gastrointestinal tract into another. In this process, a proximal bowel segment usually invaginates into an immediately subjacent distal segment, and is ordinarily associated clinically with signs and symptoms of intestinal obstruction.

This condition is observed most commonly in young children, approximately 75 per cent of all cases occurring in the age group under two. In the majority of these cases there is no demonstrable underlying pathology which may predispose to intussusception. In a series of 702 cases, Gross states that only 5 per cent had an underlying lesion, most frequently a Meckel's diverticulum, and less frequently, benign tumor or other lesion.

In contrast, it is a generally accepted opinion that intussusception in the adult usually occurs secondarily to pre-existing pathology of some type.

Most cases of intussusception are usually ileocecal or ileocolic in type. One may occasionally encounter pure enteric or pure colic forms of intussusception.

In the ileocolic form the ileum invaginates into the lumen of the right colon with the cecum remaining normally oriented; whereas, in the ileocecal form, although the ileum or the ileocecal valve spearheads the process, there is an associated invagination of the cecum and a variable length of the colon. The latter form may actually present as a mass in the rectum or be associated with prolapse of the intussuscepting segment at the anal orifice.

Clinically, the classic case of intussusception is easy to recognize in infants and children; however, the same is not true of the adult patient, especially when early or minimal degrees of ileocecal intussusception exist.

In the adult, ileocecal intussusception will not always produce a clear cut clinical picture of intestinal obstruction, nor will it always produce the classic roentgen pattern on the barium enema study which is ordinarily described in the literature.

We recently had the experience of studying 4 cases of intussusception occurring in the ileocecal area in adult patients with considerable variability demonstrated in the appearance of this condition on barium enema studies. This prompted us to review the case records of patients in whom this diagnosis was established by roentgenographic methods or at the time of surgery. The roentgenograms of 8 cases were available for review.

Clinically, these cases were broken down into two major groups. The first consisted of cases in which the intussusception was associated with fairly clear evidence of intestinal obstruction and in which an underlying pathologic lesion was discovered in the terminal ileal segment at the time of surgery. Undoubtedly, the obstructive manifestations in these cases were due to a combination of circumstances: (1) pathologic narrowing of the small bowel; (2) the factor of prolapse or intussusception; (3) very likely edema occurring in the wall of the intussuscepted segment as the result of variable degrees of incarceration. In none of these cases was the terminal ileum filled at the barium enema study.

The second group consisted of 4 recent cases in which vague symptoms consisting primarily of minimal, localized, cramping or colicky pain in the right lower quadrant were noted. It was our experience in these cases that the terminal ileum was filled at the barium enema study, and that the defect produced as a result of intussusception was quite variable in appearance. In 1 of these cases the presence of a polypoid tumor mass at the cecal base was suspected since this defect was reduplicated on two

successive barium enema studies, and on each occasion there was filling of the ileum by reflux.

ROENTGEN CONSIDERATIONS

Generally speaking, in instances of minimal protrusion or intussusception of the terminal ileum into the cecum when the ileocecal valve is normally situated (posteromedial aspect of the cecum), one will ordinarily see a cup-shaped deformity or filling defect which at times may be difficult to differentiate from a hypertrophied valve in this area. Fleishner and Bernstein[7] stated that they do not believe it is possible to distinguish between a structurally plump lid and prolapse of ileal mucosa unless the filling defect changes in size or shape during one examination, or on repeated examinations, which would be in favor of prolapse. As the defect becomes larger, the diagnosis becomes relatively simple. With continued invagination of the ileum, the cecum becomes shortened and its base begins to invaginate producing a meniscus or mushroom-shaped defect at the barium filled termination of the caput caeci.

We have noted a tendency towards spasm in the cecal area in case of ileocecal intussusception, especially in those cases with associated organic lesions in the terminal ileum; this is not, however, a constant finding.

The anatomic location of the defect in the barium filled colon due to intussusception, whether occurring medially or at the cecal base, very likely is correlated best to the degree and extent to which the ileum has intussuscepted, and whether or not the cecum itself participates in the intussusception.

In some instances, however, the location of such defects at the cecal base or posterolaterally may be due to an anomalous location of the ileocecal valve structure.[2,7,9,12]

Filling defects of the latter type occurring during barium enema study of the large bowel may erroneously lead to pro visional diagnosis of tumor, polyp or mass in the cecal area, or may casually be dismissed as due to inversion of the appendiceal stump secondary to surgery, especially when the terminal ileum has been filled by reflux. In cases of this type the proper diagnosis may be indicated only by careful study of the postevacuation enema film which may reveal disappearance of the defect previously noted, a normal outline of the cecal base, and perhaps result in filling of the appendix which was not previously outlined. With reduction of the intussusception, one may see a change in position of the cecal base, with the film showing reduction of the intussusception outlining the cecum at a lower level.

Occasionally, a progress roentgenogram made in the course of the study of the upper gastrointestinal tract may demonstrate normal findings in the ileocecal area and thus indicate the nature of a cecal defect demonstrated on barium study of the colon. Conversely, it is entirely possible that such a roentgenogram may demonstrate an intussuscepting lesion in the ileocecal area, which may have been inadvertently reduced during barium enema study of the colon.

In one of our cases, ileocecal intussusception was diagnosed only on the postevacuation study which demonstrated a mushroom-shaped defect in the cecal base with the terminal ileum clearly outlined by barium as it entered this defect from below. Incidentally, in this case, shortening of the right colon resulted from the intussuscepting process with a change in position of the barium-filled cecum being noted.

REPORT OF CASES

Case 1. N. D., a four year old white girl, was admitted on April 26, 1955, with intermittent cramping abdominal pain, nausea and passage of several loose stools. Approximately two days previously the child had noticed minimal cramping abdominal pain.

Physical examination revealed no significant findings. Urinalysis and blood cell counts revealed normal findings. Clinical impression at this time was mesenteric adenitis or appendicitis.

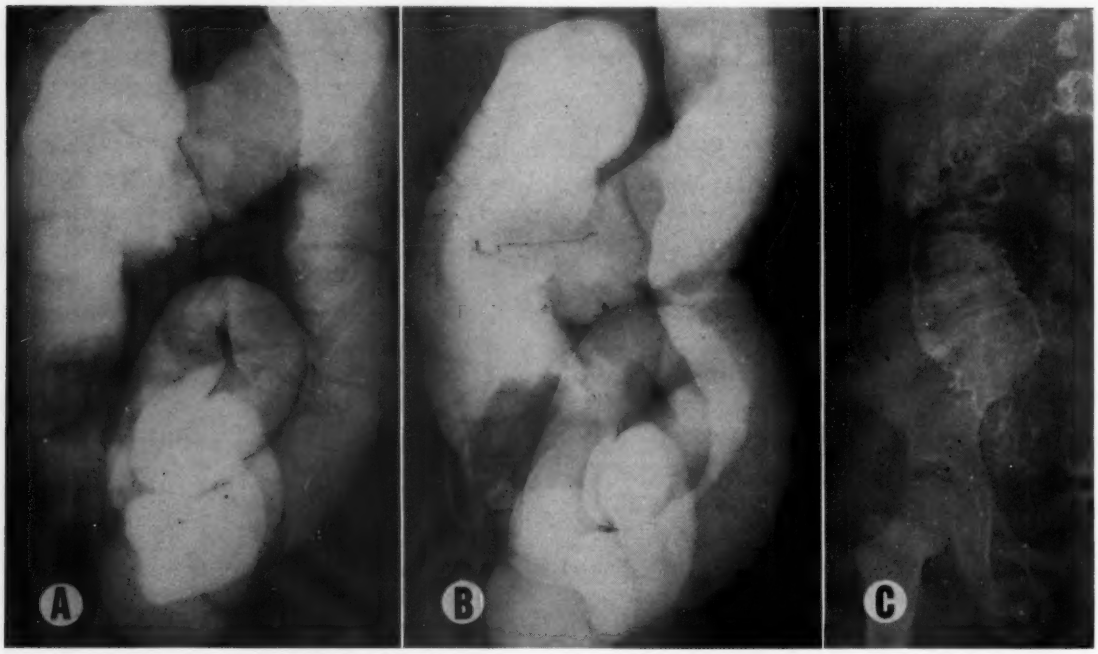

Fig. 1. Case 1. (*A*, *B* and *C*) Barium enema study in young child showing typical defect in ileocecal area due to ileocecal intussusception. Note characteristic "coiled spring or concertina effect" of mucosa of right colon on postevacuation study (*C*) indicating presence of intussusception.

Roentgen Examination. On April 30, 1955, barium enema study revealed an obstruction at the hepatic flexure which gradually shifted until the cecum was filled. A persistent large radiolucent defect at the ileocecal area was interpreted as due to incomplete reduction of an ileocecal intussusception (Fig. 1, *A*, *B* and *C*). May 1, 1955, an upper gastrointestinal study revealed barium in the terminal ileum as it entered the cecum, also a large radiolucent defect was noted in this area which was due to persistence of intussusception (Fig. 1*D*).

On May 4, 1955 the child was operated upon and an ileocecal intussusception easily reduced. The terminal ileum was thickened and edematous and would readily slip back and forth through the valve without difficulty; therefore, following reduction of the intussusception, the ileum was fixed along the medial border of the cecum and ascending colon by several sutures. The appendix was subacutely inflamed and was removed.

Fig. 1. Case 1. (*D*) Small bowel study demonstrates large polypoid defect due to ileocecal intussusception. Observe the ileum as it enters the central portion of the defect.

Comment. This case is included to illustrate the typical appearance of a defect in the ileocecal area due to intussusception. The obstructive process was partially reduced during the course of the barium enema study with intussusception recurring after expulsion of the enema as manifested by study of the postevacuation film which demonstrated a typical "coiled spring or concertina effect" and extension of the defect toward the hepatic flexure (Fig. 1*C*).

It is also interesting that the upper gastrointestinal study on the following day demonstrated the prominent defect in the ileocecal area due to ileocecal intussusception without evidence of dilatation of the small bowel in spite of a partial, incomplete obstruction since the progress films showed delayed emptying of the small bowel.

Case II. H. T., a fifty-eight year old white female, was admitted to Grace Hospital on June 12, 1955, with a chief complaint of rectal bleeding. The patient first noticed bright red blood about six months previously. At first only a small amount of blood was seen on the toilet tissue. On several occasions since she has passed one or two tablespoons of blood. There has been no pain or change in bowel habits. Patient experienced a weight loss of fifteen pounds over the past eight months, since the time of surgery for removal of a gallbladder which showed evidence of cholecystitis and cholelithiasis.

Roentgen studies revealed a small polypoid lesion, approximately 7 mm. in size in the mid-sigmoid colon and a large polypoid defect at the lateral border of the cecum thought to be due to an ectopic hypertrophied ileocecal valve, possibly with ileal prolapse (Fig. 3, *A*, *B* and *C*).

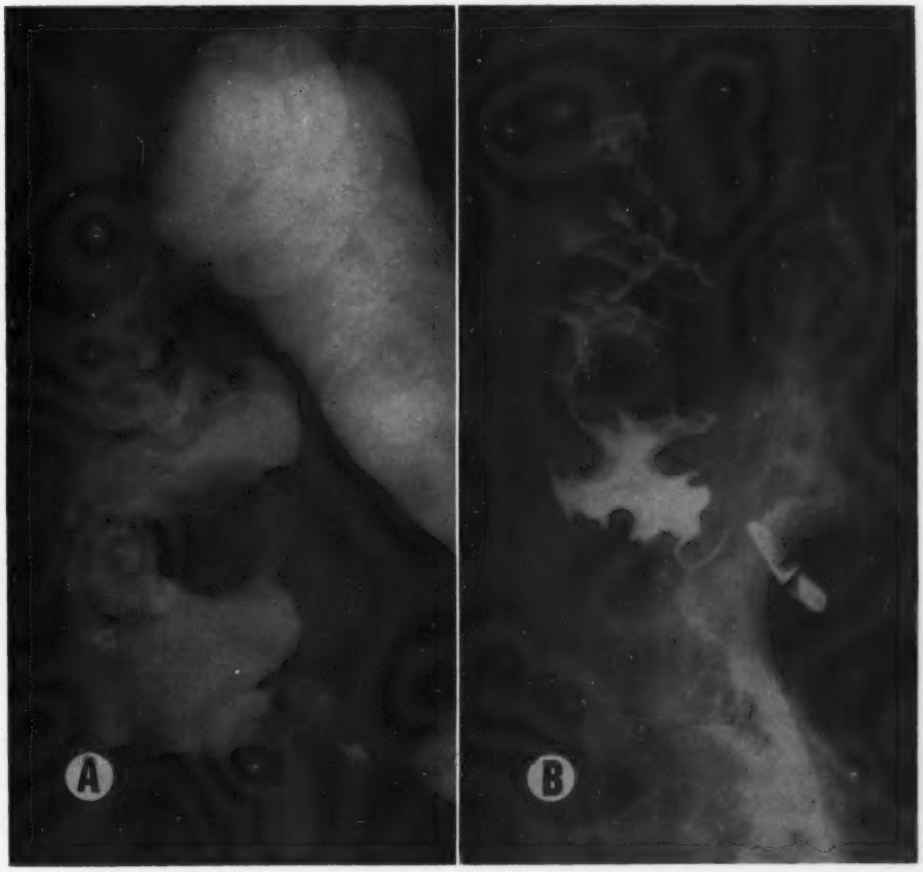

Fig. 2. (*A* and *B*) Pre- and postevacuation barium enema study demonstrating hypertrophied ileocecal valve in asymptomatic patient.

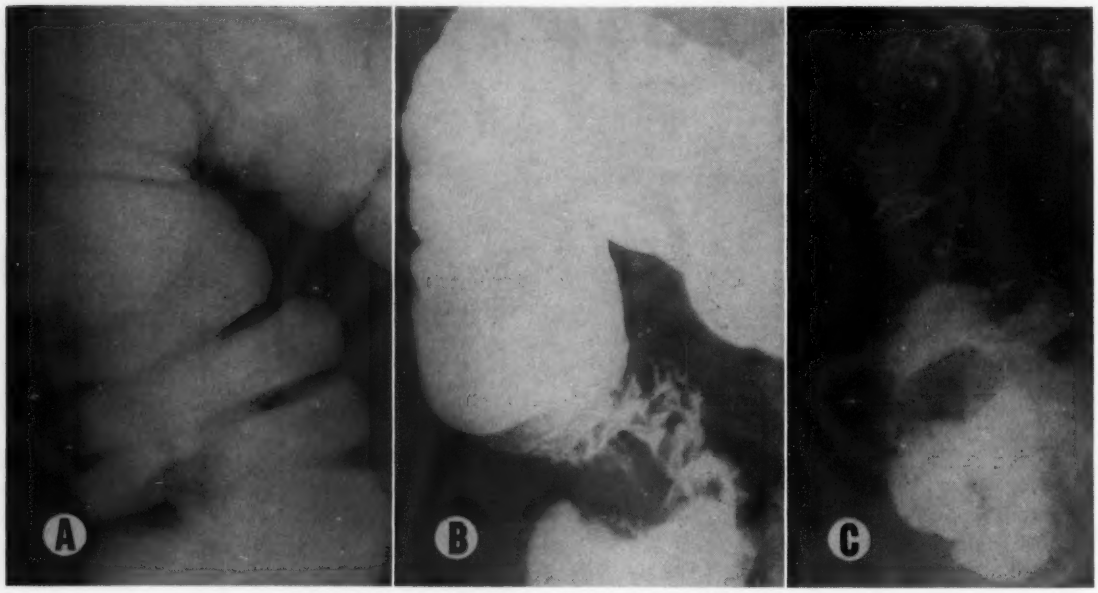

Fig. 3. Case II. (*A*, *B* and *C*) Pre- and postevacuation barium enema study demonstrating filling defect at the medial aspect of cecum due to ectopic hypertrophied ileocecal valve with ileal prolapse (surgery).

Surgery on June 17, 1955 confirmed the presence of a polyp in the sigmoid colon. In addition, a second, smaller lesion of 3 to 4 mm. in size was detected in the lower descending colon. A mass was palpated in the cecal area and cecotomy revealed a prominent, hypertrophied, ectopically situated ileocecal valve with associated ileal prolapse or intussusception. Since the ileocecal prolapse could not be aggravated by manipulation at the time of surgery,

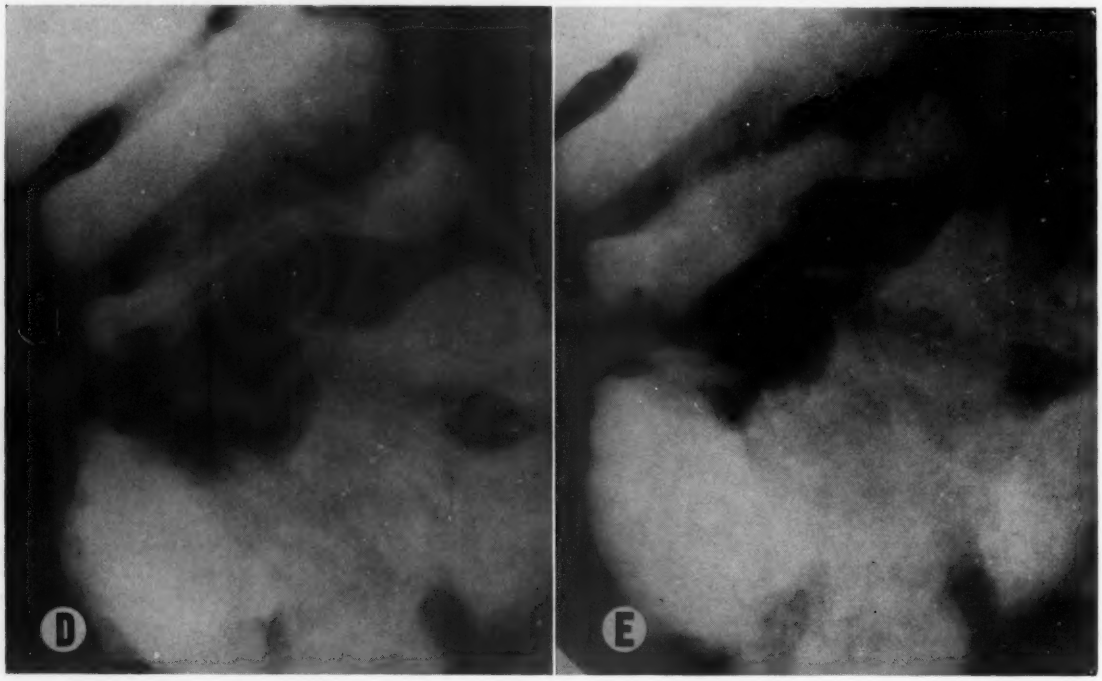

Fig. 3. Case II. (*D* and *E*) Spot compression studies of defect in cecal area. Note slit-like accumulation of barium in central portion of filling defect, conclusively identifying structure as ileocecal valve.

and since no symptoms apparently resulted from its presence, it was felt that no treatment was justified for this lesion. The patient recovered uneventfully and was discharged on June 24, 1955.

Comment. Only the compression roentgenograms permit an accurate identification of the polypoid defect in the cecal area as an ectopically situated hypertrophied ileocecal valve (Fig. 3, *D* and *E*). Obviously an accurate interpretation is necessary in order to avoid unnecessary surgery for defects of this type (Fig. 3*F*).

Case III. R. M., forty-six year old white male, was admitted on September 15, 1954, with chief complaints of increasing constipation, diarrhea, abdominal cramping and a forty

Fig. 3. Case II. (*F*) Air contrast study outlines polypoid defect—does not permit accurate differentiation of nature of this defect.

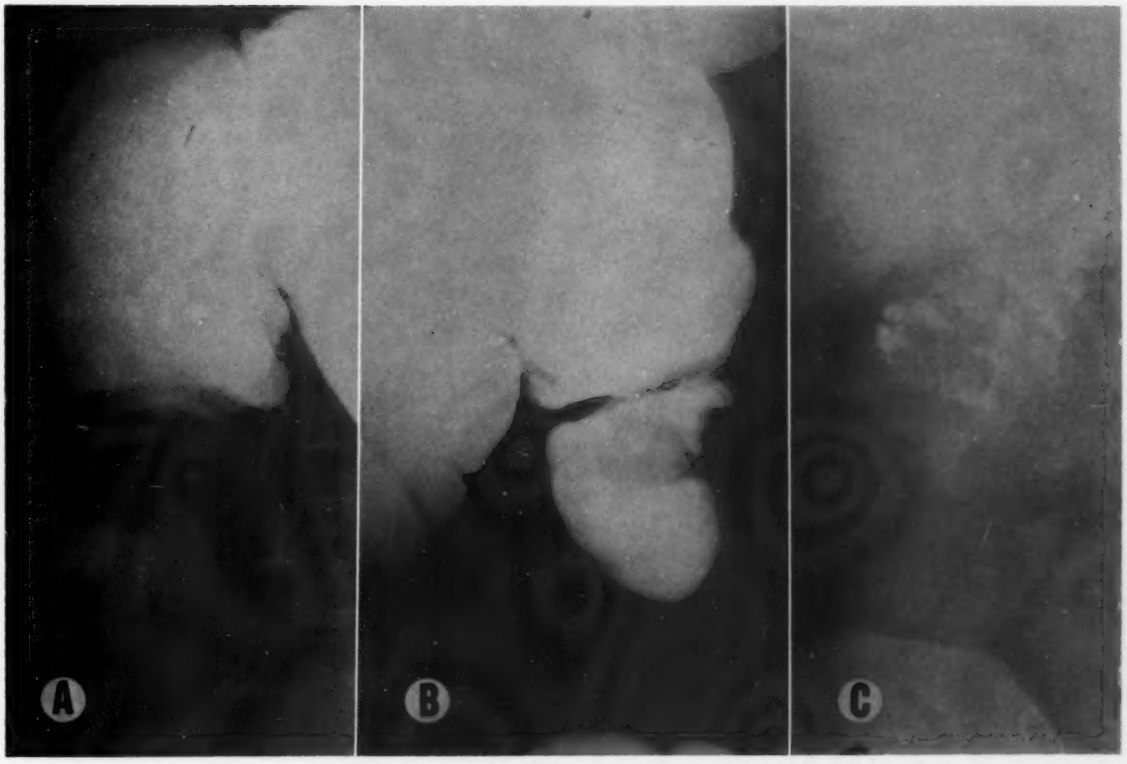

Fig. 4. Case III. (*A*, *B* and *C*) Note polypoid defect at medial border of the cecum due to ileocecal intussusception and small spastic cecum. Also note decrease in size of defect in (*B*) as compared with (*A*) due to partial reduction of intussusception. On postevacuation film (*C*) note high position of cecal base and filling defect at cecal base indicating progression of intussusception.

pound weight loss in the last six months. The patient experienced a similar attack of diarrhea and abdominal cramps six years previously which lasted two weeks and cleared completely. During the past month the patient noticed frequent abdominal cramps, gurgling sounds in the abdomen and passage of considerable mucus and bile in the stool. Tarry stools were passed on several occasions in the last month. One episode of vomiting was noted a week ago. Physical examination revealed the abdomen to be distended, hyperactive bowel sounds were audible and peristaltic waves were visible on the abdominal wall. Bilateral inguinal hernias were present.

Roentgen Examination. On September 28, 1948, upper gastrointestinal study demonstrated marked hypermotility with the barium meal reaching the rectum at two hours. No pathology was noted in upper gastrointestinal segments. Cecum and terminal ileum were unsatisfactorily outlined. On September 29, 1948, barium enema study revealed a small spastic cecum with a prominent polypoid defect at the medial aspect of the cecal base, which was interpreted as a hypertrophied ileocecal valve (Fig. 4, *E* and *F*). On September 13, 1954 barium enema examination demonstrated a large polypoid defect in the ileocecal area which decreased slightly in size as the enema filled this area (Fig. 4, *A*, *B* and *C*). The cecum was small and spastic. The terminal ileum could not be filled. A moderate gaseous distention of the small bowel was present. On September 16, 1954, after a long tube had been successfully passed, a barium meal was administered which demonstrated a marked dilatation of the entire small bowel. A very severe dilatation of the terminal ileum which measured approximately $3\frac{1}{2}$ inches in diameter was noted on the four-hour study with an annular stenosing defect demonstrated at the distal aspect of this grossly dilated ileal segment (Fig. 4*D*). Small amounts of barium entered the colon. The roentgenologic impression was (1) neoplasm of terminal ileum with very high grade obstruction and (2) ileocecal intussusception.

Exploratory surgery on September 20, 1954, revealed a hard mass involving the distal ileum and the cecal base, many nodes in the mesentery and nodules in the liver. The mass intussuscepted slightly into the cecum. The treatment consisted of right colectomy and ileo-transverse colostomy. Pathologic diagnosis was carcinoid which involved the terminal ileum, 2 cm. proximal to the ileocecal valve with a mass measuring 3.5 by 3 by 2 cm. occupying half of the circumference of the bowel lumen. The microscopic study also revealed evidence of carcinoid in the wall of the appendix and in the adjacent lymphnodes.

Comment. Several interesting speculations exist when one reviews this case history. Was the pathologic process present in 1948? Certainly the pathologic characteristics of carcinoid are consistent with a long standing history such as is present in this case. Was the defect in the ileocecal area due to hypertrophy of the ileocecal valve or was intussusception present at that time? Certainly one cannot ordinarily make this differentiation unless the defect in the ileocecal area shows change in size or shape during one or more examinations. The presence of moderate dilatation of the terminal small bowel in relation to this prominent defect, especially in the presence of a history suggesting a partial intestinal

Fig. 4. Case III. (*D*) Small bowel study outlines annular stenosing defect in terminal ileum due to involvement by carcinoid and severe dilatation of the proximal small bowel.

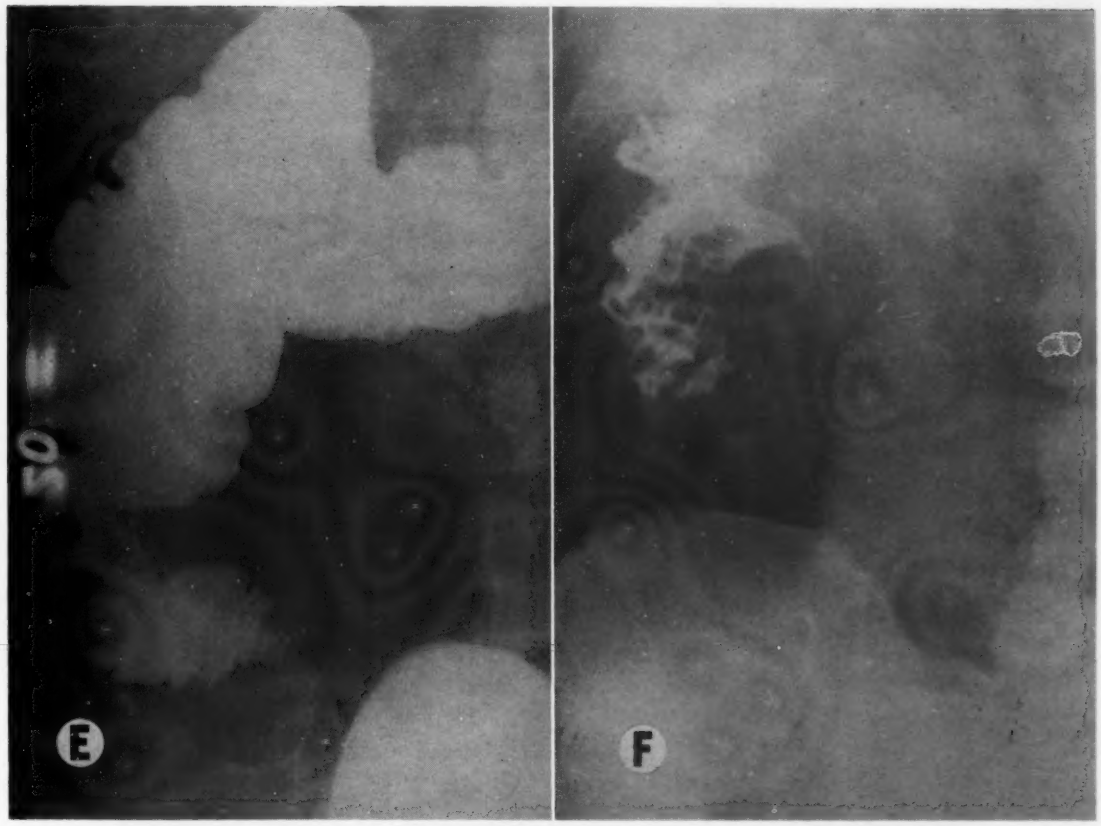

Fig. 4. Case III. (*E* and *F*) Barium enema study on September 29, 1948, demonstrated a large polypoid defect in ileocecal area interpreted as hypertrophied ileocecal valve. Note gas-distended dilated terminal ileum entering valve area. Also observe small spastic cecum.

obstruction, merits a detailed investigation of the small bowel to exclude the possibility of minimal degrees of intussusception. Does the presence of a small, spastic cecum bear special significance in the presence of unusually prominent defects in the ileocecal valve area (Fig. 5)? We have noted a tendency towards spasm in the cecal area with several of our cases of intussusception in this area; however, this has not been a constant finding. It may well be that in the study of such cases special small bowel examinations for investigation of terminal ileum and ileocecal area are justified.

Case IV. J. K., a thirty-six year old white female, was admitted on February 20, 1952, with a chief complaint of intermittent attacks of "soreness" in the right lower quadrant. This discomfort was accentuated when the patient was upright, usually lasted two to three hours, and was not accompanied by nausea or vomiting. Physical examination revealed mild tenderness in the right lower quadrant. Exploratory laparotomy was performed on February 21, 1952, and the appendix and right tube were removed. The pathologic report indicated a mild chronic appendicitis and mild chronic salpingitis.

The patient was re-admitted on May 13, 1953, with complaints of abdominal distress localized to the right lower quadrant, unrelieved by the appendectomy performed one year previously. In addition, the patient complained of slight upper abdominal distress appearing one and one-half to two hours after meals, relieved by alkalis and vomiting. The patient also experienced intermittent attacks of diarrhea.

Roentgenographic studies revealed a normally functioning gallbladder and no pathology in the upper gastrointestinal tract. The barium enema examination demonstrated an ileocecal intussusception which was reduced fol-

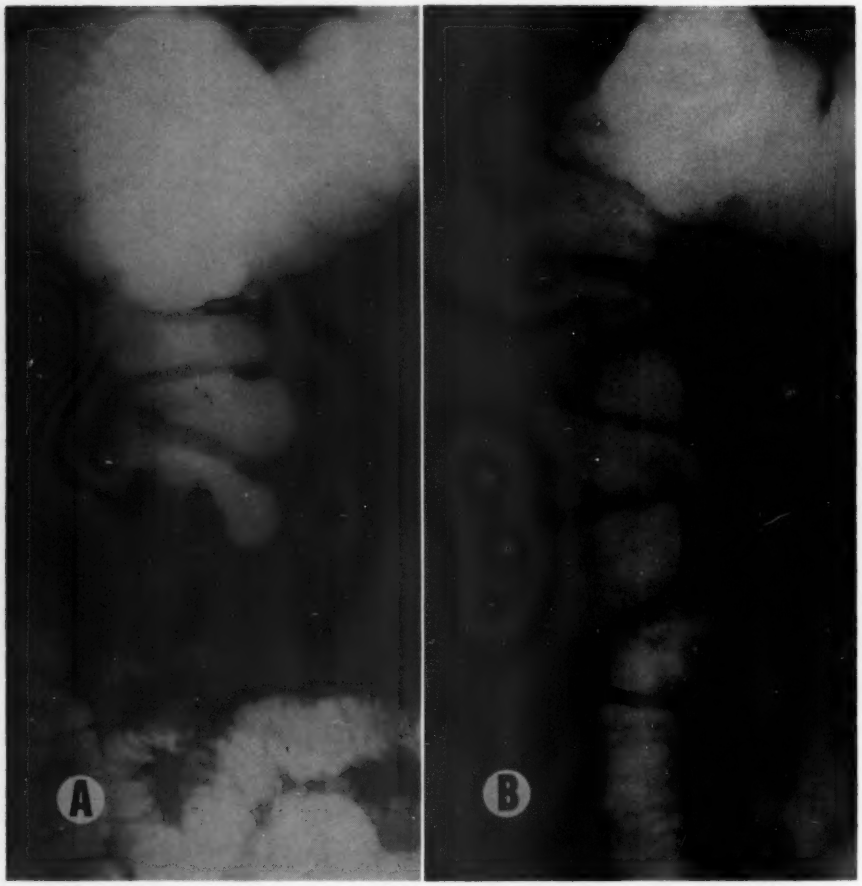

Fig. 5. (*A*) Note filling defect at cecal base visualized on right anterior oblique study and the ileum apparently entering this defect from below. Cecal spasm or cecal intussusception? (*B*) Anteroposterior roentgenogram made a few seconds later demonstrates normal findings in this area. Patient was asymptomatic.

lowing expulsion of the enema (Fig. 6, *A*, *B* and *C*). The patient was discharged without additional study.

Comment. This case illustrates very excellently one of the classic appearances of ileocecal intussusception. Note the mushroom shaped defect at the cecal base and the barium-filled terminal ileum entering this defect from below (Fig. 6, *A* and *B*). The intussusception was reduced on expulsion of the enema, the postevacuation roentgenogram demonstrating disappearance of the defect previously noted. The cecal base was in a lower position and the ileocecal valve was normally located (Fig. 6*C*).

Case v. S. M. B., a forty year old white female, was admitted on June 29, 1955, for diagnosis and treatment of a lump in the left breast. Since the patient gave a history of recurrent abdominal pain usually in the upper abdominal quadrants, recurring attacks of diarrhea and alternating constipation for several years, a complete investigation of the gastrointestinal tract was deemed advisable.

Roentgen studies of the chest, gallbladder and upper gastrointestinal tract were negative. Barium enema studies on July 5, 1955 and July 6, 1955 demonstrated a persistent filling defect at the cecal base, thought to represent a possible neoplastic lesion (Fig. 7*A*).

On July 7, 1955 a small cyst was removed from the left breast.

On July 13, 1955, following excision of an old appendectomy scar in the right lower quadrant, exploratory laparotomy revealed the presence of a polypoid-like mass at the cecal base on palpation. The cecum was opened and a marked

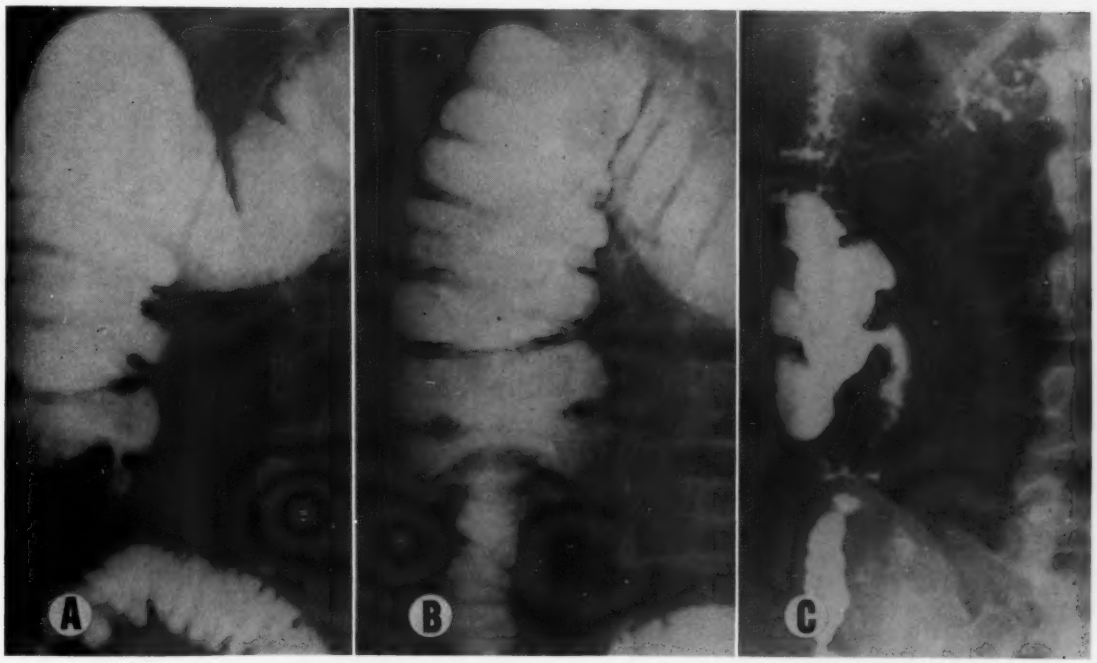

Fig. 6. Case iv. (*A*) Note mushroom-shaped defect at cecal base and barium-filled terminal ileum as it enters this defect from below. (*B*) Right anterior oblique study also demonstrates cecal defect due to ileocecal intussusception and abnormal position of the terminal ileum as it enters the cecal base. (*C*) Postevacuation film shows normal filling of the cecal base and terminal ileum entering the cecum in a normal position indicating spontaneous reduction of the intussusception on expulsion of the enema.

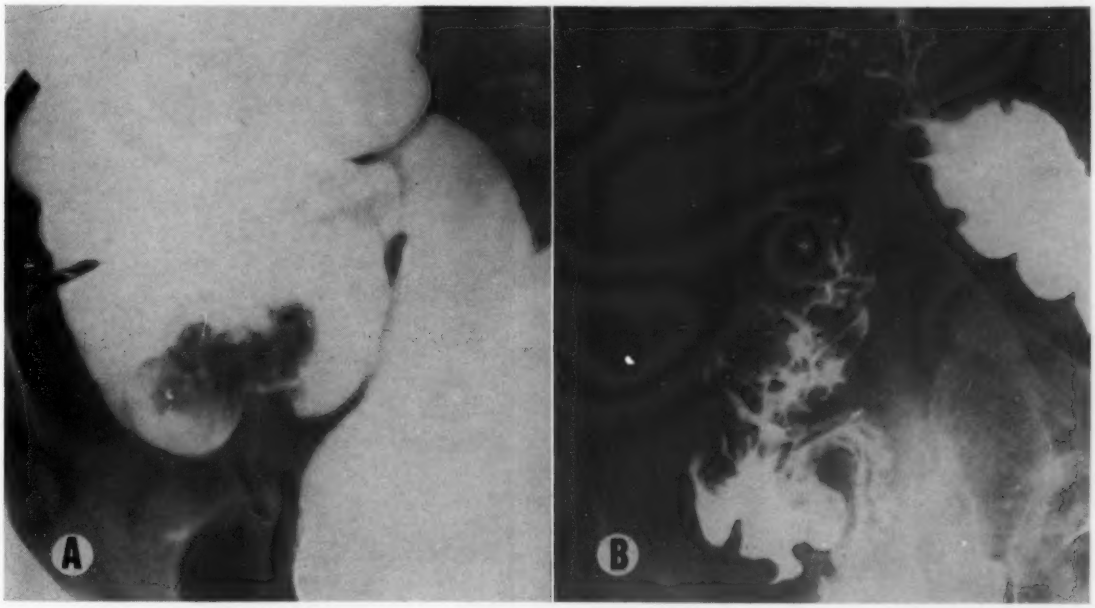

Fig. 7. Case v. (*A*) Note polypoid filling defect at cecal base on spot compression study. (This defect was reduplicated on two studies on consecutive days). Also note the apparent normal insertion of the terminal ileum. (*B*) Note disappearance of defect at cecal base on the postevacuation study and observe partial filling of the appendix indicating reduction of intussusception.

prolapse of the mucosa of the terminal ileum was found. It was felt that this explained the findings noted on palpation and discovered on roentgen examination. The intussusception was reduced; however, no definitive treatment was carried out. It was felt that in this case there was an ectopic location of the ileocecal valve laterally and posteriorly as well as somewhat inferiorly. A small subserous uterine myoma was also excised at this time. The postoperative course was uncomplicated and the patient was discharged on July 25, 1955.

Comment. Note the polypoid filling defect at the cecal base (Fig. 7*A*). This finding was demonstrated on barium enema studies carried out on two successive days. Each time the terminal ileum was outlined. It was felt that this defect was probably due to a polypoid tumor mass in this area. Note on the postevacuation roentgenogram a normal filling of the terminal ileum, cecal base and a partial filling of the appendiceal stump (Fig. 7*B*). A correlation of the findings on the pre- and postevacuation studies should have indicated the presence of an intussuscepting process at the cecal base. One could not differentiate roentgenographically in this case, a minimal ileocecal intussusception (as established at the time of surgery) from a minimal intussusception of the appendiceal base. The defect appears too large and somewhat irregular. As such, simple inversion of the appendiceal stump secondary to surgery appears unlikely.

Case vi. R. R., a fifty-nine year old white male, was admitted on September 30, 1952, with a chief complaint of cramping right lower quadrant pain of five days' duration associated with nausea and vomiting. The patient had also noted hematuria on several occasions. The past history was noncontributory. No history of previous surgery was obtained. Physical examination revealed moderate distention of the abdomen, hyperactive bowel sounds on auscultation, and minimal tenderness in the right lower quadrant. The clinical impression at this time was intestinal obstruction.

Roentgen Examination. On October 1, 1952,

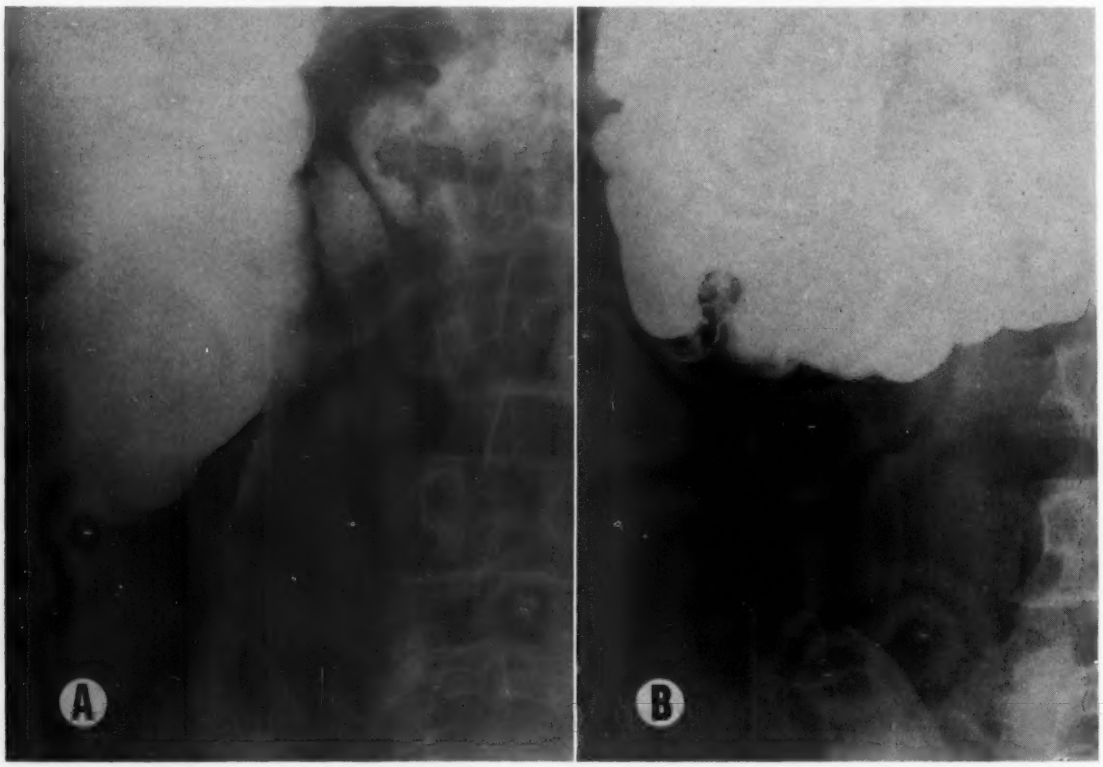

Fig. 8. Case vi. (*A*) Pre-evacuation barium enema study demonstrates normal findings in the ileocecal area. Observe gaseous distention of small bowel loops. (*B*) Observe the high position of the cecum on the postevacuation study and the mushroom-shaped defect. The terminal ileum enters from below.

cystoscopic and pyelographic studies revealed no abnormality. On October 3, 1952, upper gastrointestinal and small bowel studies showed delay in passage of barium as it reached the terminal ileum. No significant dilatation of small bowel loops was seen. A 24-hour film demonstrated small amounts of barium remaining in the distal small bowel. Roentgenologic impression at this time was partial small bowel obstruction, cause undetermined. On October 4, 1952, barium enema study revealed a normal filling of the colon and terminal ileum without evidence of pathology (Fig. 8*A*). The postevacuation film demonstrated evidence of intussusception at the cecal base (Fig. 8*B*). On October 7, 1952, a flat roentgenogram of the abdomen demonstrated retention of barium in the distal small bowel and an outline of the right colon. A long tube had been successfully passed (Fig. 8*C*).

On October 8, 1952, exploratory laparotomy revealed a congenital band in the right lower quadrant which produced a kinking of the terminal ileum and intussusception of the terminal ileum into the cecum. The band was severed and the intussusception reduced.

Comment. It is interesting to note that the only specific evidence of intussusception was demonstrated on the postevacuation study (Fig. 8*B*). Note the small mushroom-shaped radiolucent defect at the cecal base with the terminal ileum entering this defect from below. Note also the shortening of the right colon with ascent of the cecum as intussusception occurred. One can best gauge this change by comparison with a fixed point, *i.e.*, the iliac crest. It is entirely possible that the intussusception in this case was inadvertently reduced as barium filled the cecal area.

Case vii. G. G., a sixty-one year old white female, was admitted to the hospital on July 11, 1955, for investigation of a polypoid defect demonstrated at the base of the cecum on a routine barium enema study (Fig. 9, *A* and *B*). The interpretation of this defect on the original examination was a probable invagination or inversion of the appendiceal stump secondary to surgery of the appendix. On reviewing the case with the attending physician it was revealed that the patient had never experienced abdominal surgery of any type; therefore, a reexamination of the bowel was requested. It was suspected that this defect might be due to a

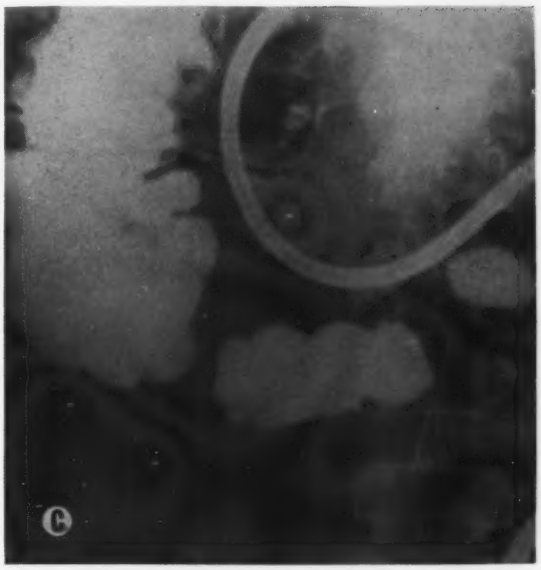

Fig. 8. Case vi. (*C*) A 72-hour film of the abdomen demonstrates retention of barium in the terminal ileum as well as in the right colon.

minimal intussusception of the terminal ileum, or possibly, to intussusception of the appendix. The patient's only complaints were that she experienced an occasional low back pain at the base of the spine which radiated slightly to each side, more so on the right. There were no complaints referable to the gastrointestinal tract.

On the repeat barium enema study, the cecal base was normally filled (Fig. 9*C*). The defect previously observed was not present and a thin normal appearing appendix was outlined with barium. It was therefore assumed that the original defect was either due to intussusception of the appendix into the cecum or we were dealing with a minimal intussusception of the ileum, possibly due to an anomalously situated ileocecal valve, which produced a minimal partial invagination of the cecal base with the appendix participating secondarily in this process. Surgery was not deemed advisable at this time in view of the absence of clinical findings and the patient was discharged.

Comment. Fraser[8] has written an excellent paper dealing with the subject of appendiceal intussusception. The reader is referred to his paper for a résumé of this subject.

Case viii. L. M., a forty-two year old white male, was admitted on July 8, 1954, with a chief complaint of a mass in the right lower quadrant and intermittent colicky right lower quadrant

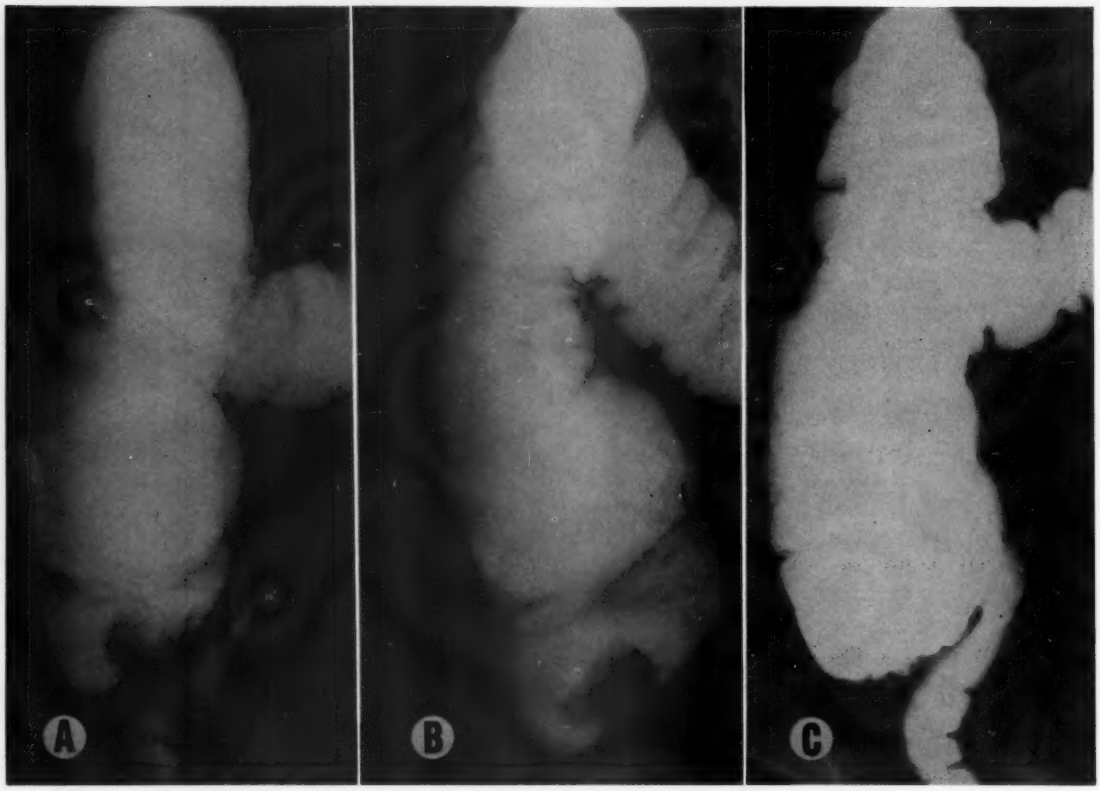

Fig. 9. Case VII. (*A* and *B*) Note polypoid defect at cecal base due to intussusception of appendix. (*C*) A repeat barium enema study one week later demonstrates normal filling of the cecal base and outlines a somewhat thin atrophic appearing appendix.

pain. Otherwise the patient was asymptomatic. Physical examination revealed an indefinitely outlined mass in the cecal area which was nontender, but was otherwise negative. Laboratory examinations which included urinalysis and blood studies, as well as studies of stool for parasites, were completely negative.

Roentgen Examination. The barium enema study demonstrated a polypoid defect at the medial border of the cecum interpreted as either due to a hypertrophied ileocecal valve or possibly to a minimal degree of ileal prolapse or intussusception (Fig. 10*A*). The upper intestinal and small bowel studies demonstrated a pathologic narrowing of the terminal ileal segment as it entered the ileocecal valve area with a complete loss of mucosal pattern in this segment (Fig. 10*B*). At the time of the small bowel study, air was insufflated into the colon which outlined the ileocecal area and, in addition to the defect at the ileocecal valve previously noted on the barium enema study, revealed evidence of a fairly large, extrinsic pressure defect at the medial border of the cecum (Fig. 10*C*). The roentgen interpretation at this time was a regional enteritis or Crohn's disease involving the distal ileum with associated granulomatous mass in the ileocecal area and possibly minimal ileocecal intussusception.

Surgery on July 14, 1954 confirmed the presence of a mass located in the cecal wall producing extrinsic pressure on the terminal ileal segment. The appendix could not be identified. The terminal ileum and cecum were resected *en bloc* and gross study of these structures demonstrated in addition a definite intussusception of the ileum into the ileocecal valve area. The mass involved the ileum only secondarily by pressure, and no intrinsic alteration of the terminal ileal segment was noted. Histologic study of the tissue revealed a nonspecific granulomatous mass, apparently due to the perforation of the appendix. The patient recovered uneventfully and was discharged.

Comment. Of interest in this case is the defect in the ileocecal valve area (Fig.

10*A*) which one would be justified in assuming to be due to a hypertrophied valve, unless one could show evidence of change in the size or shape of the defect on serial study. The latter finding would then indicate the possibility of minimal ileocecal intussusception. Again of interest in this case is the fact that a fairly large mass in the cecal area was completely obscured on the barium enema study and outlined only after insufflation of air into this segment, accurately indicating that there was a mass-producing extrinsic pressure on the cecal base (Fig. 10*C*).

Case ix. L. H., sixty-six year old white male, was admitted on March 28, 1950, with a chief complaint of periumbilical pain and vomiting. On March 5, 1950, the patient noticed the onset of periumbilical pain of short duration which occurred intermittently throughout the entire month along with vomiting of brown and greenish material. On several occasions the patient vomited dark or "coffee ground like" material. The patient noted a weight loss of twenty pounds since the onset of the present illness. On October 22, 1948, the patient was operated upon for an acute purulent appendicitis with an uneventful recovery. Otherwise, the past history was noncontributory. Physical examination revealed the abdomen to be distended and tympanitic. An old well-healed scar was seen in the right lower quadrant.

Roentgen Examination. Barium enema study on March 29, 1950 revealed a localized deformity of the cecal base, and a small radiolucent defect was noted at the central portion of the caput caeci on the posteroanterior film (Fig. 11, *A* and *B*). The terminal ileum could not be filled. Considerable gaseous distention of the small bowel was noted and an obstructive process in the ileocecal area indicated. A decompression tube was successfully passed and a small bowel study on April 5, 1950 revealed a complete obstruction in the terminal ileum with no barium entering the colon at the 24 hour observation.

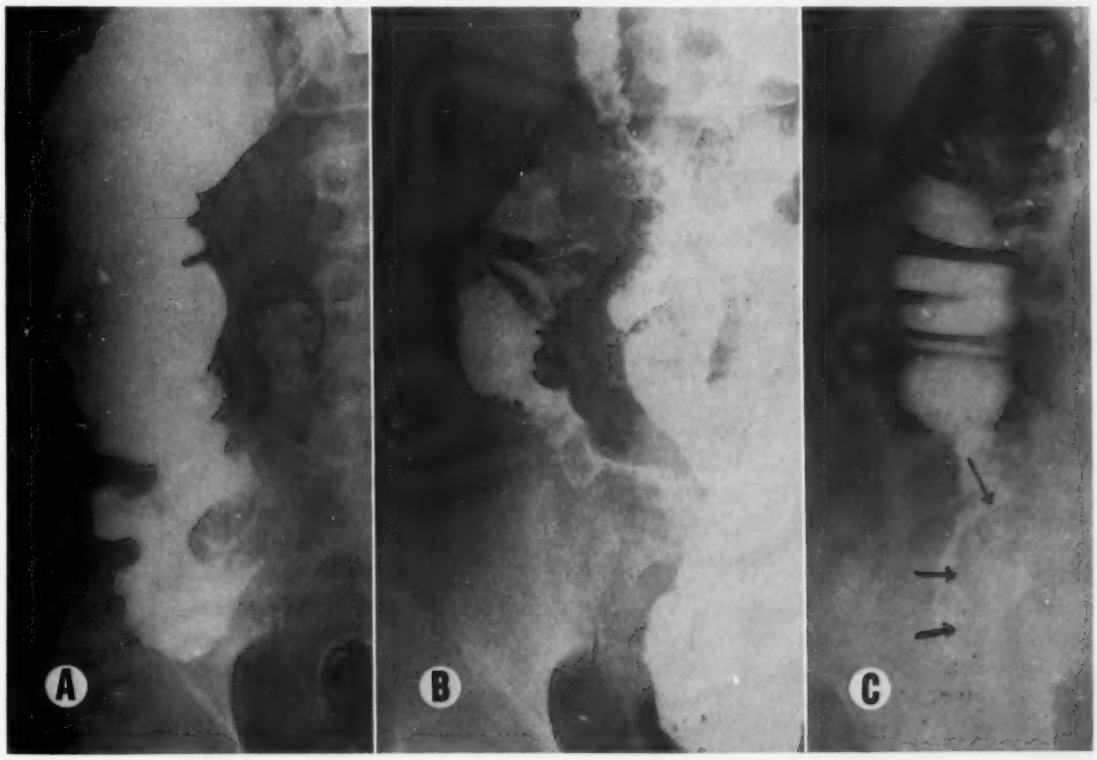

Fig. 10. Case viii. (*A*) Note the polypoid defect in the ileocecal area. (*B*) Small bowel study outlines a pathologically narrowed terminal ileum which is very irregular with the barium meal entering the right colon. (*C*) Roentgenogram made immediately after *B* following insufflation of air rectally. Arrows outline a large defect at the medial border of the cecum, plus the defect at the ileocecal area observed in *A*.

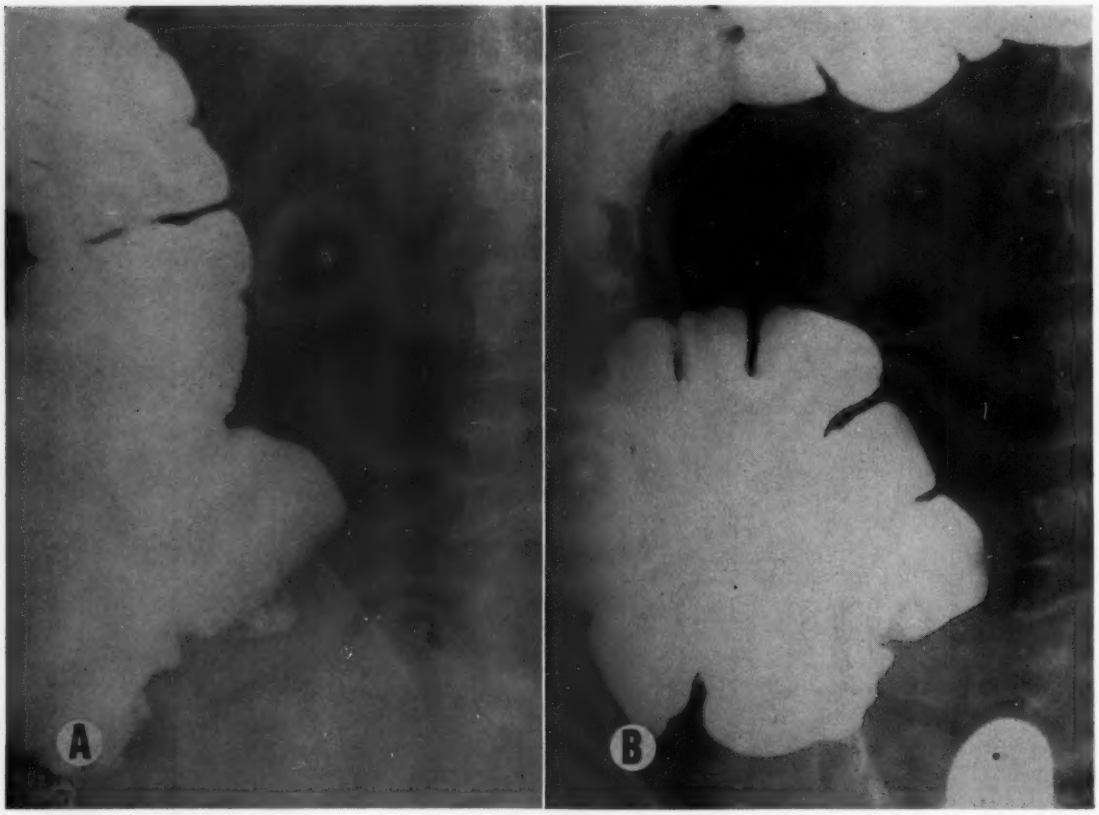

Fig. 11. Case IX. (*A*) Note bizarre defect at the cecal base and severe gaseous distention of small bowel on the anteroposterior roentgenogram. (*B*) On the posteroanterior roentgenogram the defect at the cecal base has almost completely disappeared. Note small mushroom-shaped defect at the center of the cecal base and the abnormal flattened contour of the cecal base due to ileocecal intussusception.

Exploratory laparotomy on April 13, 1950 disclosed numerous adhesions involving the small bowel. After lysis of these adhesions an annular type of tumor was found at the junction of the jejunum and ileum. This lesion including 6 inches of bowel on either side of the tumor was resected locally. A soft nodular mass was palpated in the ileocecal area. Its exact nature was not established; however, 4 inches of the terminal ileum and the cecal base were resected with the ileum subsequently being anastomosed to the transverse colon. Pathologic study revealed both lesions to be annular adenocarcinomas. The cecum was normal.

The patient suffered a partial dehiscence of the abdominal wound necessitating secondary closure; however, he recovered and was discharged on May 8, 1950.

Comment. Note the unusual defect at the cecal base with a small mushroom-like radiolucency at the central portion of the cecal base when the patient was prone. Note that the defect is also somewhat smaller on the posteroanterior film indicating partial reduction of an intussuscepting process in this area (Fig. 11, *A* and *B*).

DISCUSSION

In 1793, Hunter[13] in his paper "On Intussusception" postulated that the invagination of a contracted portion of the gut into the relaxed segment below might well be a common physiologic accident, and further, that if, for some unknown reason, this should persist so that the bowel below draws itself up on the inner segment, then an intussusception arises which grows at the expense of the ensheathing bowel.

It is generally agreed that a majority of cases of intussusception begin in the ileocecal area. It is also agreed that a majority

of cases in the adult will demonstrate an underlying exciting cause usually a benign tumor, Meckel's diverticulum, or occasionally, some underlying inflammatory process such as gastroenteritis or regional ileitis. In the presence of tumor, whether intramural or intraluminal, there is a general agreement that the normal pattern of peristaltic activity may be adequate to cause invagination of the mass into the segment of bowel immediately subjacent with resulting intussusception. In various inflammatory states, especially gastroenteritis, the pattern of peristaltic activity is accentuated and this may predispose to intussusception.

There is no satisfactory explanation for the occurrence of ileocecal intussusception in cases in which there is no demonstrable abnormality in the terminal ileal segment or in the ileocecal valve. Various theories of causation have been proposed. Blum[5] has summarized these in his paper on "Nontumid Ileo-Colic Intussusception in an Adult." We are quoting these directly from his paper.

1. That the ileocecal valve normally projects through the wall of the cecum so that during the discharge of intestinal contents it telescopes into the lumen of the large gut. When due to some derangement of intestinal rhythm this may become marked, the process may over-reach itself and an intussusception of the usual type with the ileocecal valve as the apex can be so started.

2. That the base of the appendix due to a local inflammatory process can similarly be the initial point and drag the adjacent ileocecal valve with it as the forefront of the intussusceptum.

3. That the lymphoid tissue aggregates (Peyer's patches), which are of considerable size during the first year of life and may persist in an enlarged form, serve as intramural tumors and so start the process in the last inch or two of the ileum.

4. That the slope of the mesentery, that is the angle at which the ileum joins with the cecum, may tend to precipitate invagination of the small into the large bowel, and that the reason for its predominance in the male is related to the more obtuse ileocecal angle in infants of this sex.

5. That the mesentery may be unusually long and that the peritoneum enclosing the ascending colon may be unusually lax, resulting in a mobile caput caeci.

6. Blum himself postulated that the failure of a protective sympathetic mechanism, weakest at the ileocecal junction, because of its transition from the vagal to the sacral parasympathetic, might logically explain the occurrence of the ileocecal intussusception.

SUMMARY

1. Ileocecal intussusception in the adult will not always result in intestinal obstruction, nor will it always produce constant or classic roentgen deformities on barium enema study.

2. Deformities produced by minimal degrees of ileocecal intussusception can be identified if one properly evaluates these defects on the basis of their appearance before and after expulsion of the barium enema.

3. Physiologic degrees of ileocecal intussusception may exist. Reduction of ileocecal intussusception on expulsion of a barium enema in relatively asymptomatic patients would tend to support this opinion.

4. The importance of accurate interpretation of defects in the ileocecal area due to minimal degrees of intussusception is stressed. Accurate roentgen diagnosis will eliminate unnecessary surgical procedures based on erroneous roentgen interpretation of defects in this area.

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DIVERTICULA OF THE APPENDIX*

A CASE OBSERVATION

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ALTHOUGH diverticula have been commonly observed arising from nearly every hollow viscus and constitute an especially common entity throughout the gastrointestinal tract, accounts of their presence in the vermiform appendix are few. Pathologists report incidences varying from 0.22 to 2.23 per cent.[6]

FIG. 1. Anteroposterior roentgenogram of barium-filled colon.

FIG. 2. Detail of right colon to show diverticula of appendix and cecum.

The discovery of diverticula of the appendix on roentgen examination is much less frequent. The first reported case was that of Spriggs and Marxer,[5] who, in 1926, demonstrated a diverticulum in a retrocecal appendix. Two more cases were reported in 1932, one a case of multiple appendiceal and cecal diverticula, the other a solitary diverticulum in the appendix of a patient suffering from ulcerative colitis. The first report of this condition in the American literature was by Gilmore and Mahan[2] who demonstrated diverticula in the appendix on barium enema examination of a young male adult who presented symptoms of chronic right lower quadrant pain and tenderness. Another investigator[4] described a solitary diverticulum of the appendix demonstrated on barium enema examination of a patient with ileocecal tuberculosis. Ten years later, Friedmann[1] reported a case of diverticulosis of the appendix which occurred as an incidental finding bearing no relation to the patient's clinical situation.

A case of multiple diverticula of the appendix was observed by the writers in a patient who also had a cecum containing

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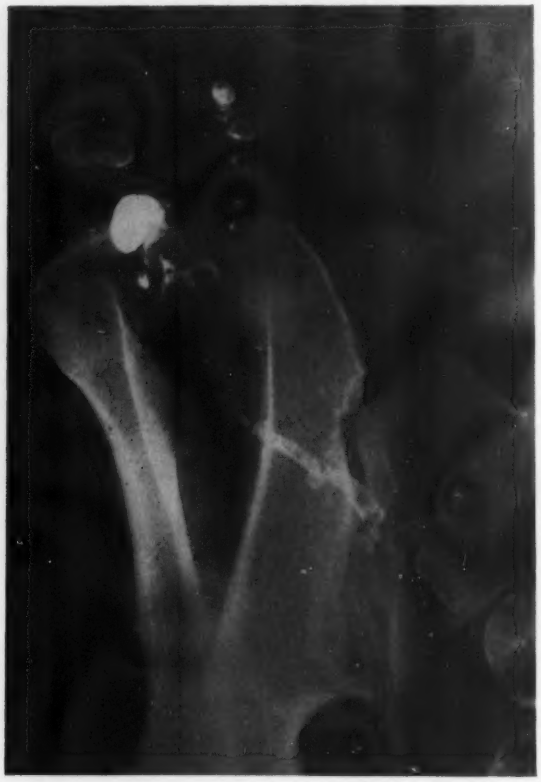

FIG. 3. Detail of postevacuation roentgenogram to show diverticula of appendix and cecum.

numerous diverticula. Considerable spasm of the cecum was encountered at fluoroscopy. The patient's only gastrointestinal complaint was bleeding from external hemorrhoids. Figures 1 and 2 demonstrate the diverticula of the appendix following barium enema study, and Figure 3 demonstrates the diverticula on a delayed postevacuation roentgenogram.

SUMMARY

A case of multiple diverticula of the appendix was observed. An account is given of 6 similar cases previously reported in the literature. In only one of the cases was right lower quadrant pain present and, therefore, it would appear that the presence of diverticula in the appendix is generally of little clinical significance.

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PNEUMATOSIS CYSTOIDES INTESTINALIS

REPORT OF THE FIRST CASE COMPLICATED BY FATAL RUPTURE OF THE COLON

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PNEUMATOSIS cystoides intestinalis, or abdominal gas cysts, was first seen in pathologic specimens over 200 years ago.[12] A disease with similar characteristics, first reported in 1825,[9] is commonly seen in hogs and rarely seen in sheep and chickens.[11] Approximately 300 cases have been reported to date. In recent years, reports have appeared with increasing frequency[3,4,6,10,13,14] since the disease may now be recognized by roentgen examination or even at sigmoidoscopy.[8] It is felt that the publication of the following case is worthwhile since no previous reference to rupture of the colon as a complication of this disorder was discovered in the literature.

REPORT OF A CASE

J. Z. (#25,957), a forty-one year old unmarried white male garbage collector, was first admitted to Ephraim McDowell Memorial Hospital, Danville, Kentucky on July 24, 1956, complaining of abdominal distention with vomiting and mild abdominal cramps for the preceding two weeks.

Although the patient had always been underweight, he felt he had been relatively healthy all of his life. He had suffered no significant past illnesses other than repeated episodes for many years of abdominal distention associated with mild distress and occasional vomiting and diarrhea. No exciting factor for these episodes could be uncovered, and they usually abated after a few hours without medication. The presenting complaint was similar to the former ones, albeit more severe and persistent. He was not given to drink, drugs or tobacco, and denied any food intolerance. No blood had been seen in his stools. There had been a few pounds of weight lost during the present illness.

A review of the systems and the family history were noncontributory.

Physical examination revealed a thin, tall man in no acute distress. He was afebrile. Blood pressure, pulse and respirations were normal. Remarkable abdominal distention was present, and peristaltic activity was easily visible through his thin abdominal wall. Occasional bursts of very loud explosive sounds were heard on abdominal auscultation. The abdomen was not tender. Neither fluid wave nor abnormal masses could be felt. The liver and spleen were not palpable. The physical examination was otherwise negative.

The blood Kahn test was nonreactive, and the urine was negative. The white blood cell count was 5,900 with a normal differential. The red blood cell count was 3,930,000, and the hemoglobin was 12.8 gm. per cent. Total plasma proteins were 5.15 mg. per cent with 3.32 mg. per cent albumin and 1.83 mg. per cent globulin.

A chest roentgenogram and intravenous urograms were within normal limits. Roentgenogram of the abdomen showed remarkable distention of the large and small bowel with multiple fluid levels in the small bowel (Fig. 1). No free air was seen in the peritoneal cavity. At the time of the roentgenographic examination, the patient declared he was much less distended than upon frequent occasions in the past. This was corroborated by worn grooves on his belt. By this time all vomiting and abdominal distress had ceased. An upper gastrointestinal and small bowel roentgen examination showed mild cardiospasm. The stomach and duodenum were intrinsically normal. All of the small bowel was dilated and contained considerable fluid. The small intestinal wall seemed slightly thickened, and its mucosal pattern was somewhat coarsened. No appreciable segmentation, intraluminal defects nor points of obstruction were noted. A few rounded radiolucent areas were noted scattered along the wall of the small bowel, particularly in the jejunum. Transit time was prolonged with five hours elapsing before barium reached the colon.

A barium enema study revealed the large bowel to be capacious but without intraluminal lesions or areas of stricture. Numerous small rounded radiolucent areas, similar to those seen in the small bowel, were noted in the wall of

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Fig. 1. Upright roentgenogram of the abdomen. The large and small intestines are distended. Multiple fluid levels are present in the small bowel.

the ascending, transverse and proximal descending colon producing a reticulated, cyst-like appearance (Fig. 2). These cysts did not compromise the lumen of the colon, and its mucosal pattern was not disturbed. A diagnosis of mild cardiospasm and pneumatosis cystoides intestinalis was made, but the nature of the small intestinal disturbance was obscure.

The patient responded fairly well to supportive therapy. He was placed on terramycin and sulfathalidine and discharged. After discharge the patient continued to lose weight and vigor and had more frequent episodes of abdominal distress and vomiting which were often associated with moderate diarrhea. These illnesses led to two further hospital admissions, and upon both occasions response to supportive treatment occurred. Studies on these admissions shed no further light on his disease, although he slowly lost about 15 pounds of weight, and his red blood cell count descended to 3,790,000 and his hemoglobin to 10.0 gm. per cent. The roentgenographic studies were not repeated.

He was last admitted on September 21, 1956, complaining of severe abdominal pain which came on suddenly without a known precipitating event just prior to hospitalization. When examined, he was in profound circulatory collapse. The abdomen was silent, and his left leg was swollen. Pain and shock were never completely overcome despite all medications. He maintained a low grade fever. On the fifth hospital day he became cyanotic, lapsed rapidly into coma and died.

An autopsy at the funeral home, limited to the abdomen, revealed numerous large gas cysts disposed subserosally along most of the small and large intestines. A small amount of fecal material was present in the abdominal cavity, and a large quantity of chocolate-colored, foul-smelling fluid was present in both gutters. A small perforation was noted at the splenic flexure. The entire small bowel was greatly distended, and its distal half was considerably reddened. No mucosal ulceration or other abnormality was noted in the region of the rupture. Gross examination of the entire large and small bowel failed to reveal a point of obstruction, tumor, ulceration, mucosal disease or other abnormality. The liver and spleen appeared grossly normal. Unfortunately, the specimens were lost in transit to the pathology department.

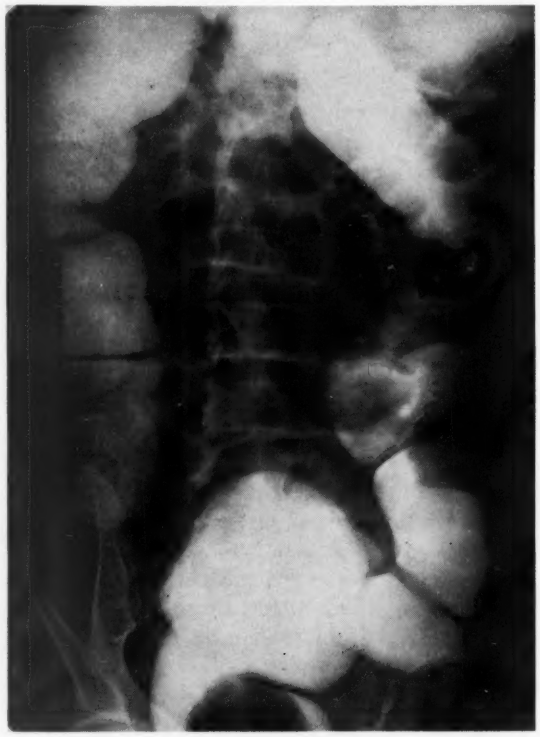

Fig. 2. Roentgenogram at barium enema examination. The colon is large. Note the reticulated, cyst-like appearance of the colonic wall, particularly in its ascending portion.

Although the patient may have had a thrombophlebitis in his left leg with pulmonary embolism, the colonic rupture with resultant peritonitis was adequate cause for death. It was felt that the tear in the splenic flexure was probably occasioned by rupture of one of the large cysts which were abundant in this area.

DISCUSSION

A comprehensive study of pneumatosis cystoides intestinalis was made fairly recently by Koss,[5] and much of the following information was taken from his article.

The disease is more common in males than females, occurring in a ratio of about 3.5:1. It is most frequently seen in infancy and middle age. In about 15 per cent of cases no associated disorder is found, being so-called "primary pneumatosis," whereas about 60 per cent of these patients have an additional pyloric obstruction, most commonly secondary to a peptic ulcer. The remaining cases suffer from various associated intestinal diseases such as a typical ulcerative colitis, regional enteritis, malignancy, etc. In infants the cysts are particularly prone to be associated with an enteritis. Marshak, Lipsay and Friedman[8] reported a case in which they felt the trauma of a sigmoidoscopy was of etiologic significance.

The cysts may be widely dispersed along the intestinal tract, although the lower small bowel and ascending colon are perhaps most commonly involved. Interspersed uninvolved intestinal segments, so-called "skip areas," are frequent. Cysts have also been reported on rare occasions in the parietal peritoneum, gastrohepatic and falciform ligaments, vaginal wall, gallbladder and retroperitoneal lymph nodes. The cysts are usually found subserosally in adults and submucosally in infants. Only minor inflammatory reaction, usually foreign body in type, may be found around the cysts which usually show an endothelial lining. The cysts may vary from microscopic size to that of a child's head.[7] No general agreement has been reached concerning the composition of the gas within the cysts. It appears to be considerably different from air, particularly in its low oxygen content.

A completely satisfactory explanation of the manner in which the gas reaches the cysts has not been forthcoming. Most students of the disease feel that the cysts represent distended lymphatics. A neoplastic theory propounded by Bang[2] has proved untenable. The bacterial theory of the origin of the cysts, which holds that the gas is formed by gas-producing microorganisms, has not met widespread acceptance. The fact that most of the patients with pneumatosis are debilitated or malnourished suggests that the gas might be produced by faulty cellular metabolism. The so-called mechanical theory postulates that the gas reaches the lymphatics through a mucosal defect. This line of reasoning is most generally accepted, although it, too, does not explain all the clinical and pathologic findings in every case.

Although the cysts may be silent and discovered accidentally, the patient usually gives evidence of his associated disease. An unusual number of cases, however, have complained of vomiting, distention, abdominal pain, weight loss and diarrhea. At times the diarrhea is bloody. Intestinal obstruction has been quite common. Besides compression or obstruction of the intestinal lumen by the cysts or secondary adhesions, the bowel containing many large cysts seems very prone to volvulus. Recurrent volvulus may account for the episodes of vomiting and pain seen in the patient reported above. Pneumoperitoneum, apparently secondary to rupture of a cyst, has been seen on many occasions, at least once with fatal issue.[3] No prior case of rupture of the bowel wall as a complication has been discovered.

Diagnosis before death or surgery is usually made by roentgen examination. At times a flat roentgenogram of the abdomen will show a double contour appearance of the bowel wall caused by gas in cysts surrounding the intestinal lumen. Occasionally, the reticulated appearance of the numerous small cysts may suggest the disorder. Of

course, spontaneous pneumoperitoneum should always arouse a suspicion of pneumatosis. The Chilaiditi sign, or interposition of a loop of bowel between the liver and the diaphragm, has been unusually frequent in these cases. The reticulated, cyst-like appearance of the wall of the bowel at barium enema examination is diagnostic. The diagnosis has also been made by sigmoidoscopy upon finding grape-like masses in the bowel wall that "popped" and collapsed on biopsy.[10]

Treatment directed toward the disease associated with the pneumatosis should always be instituted. Proponents of the bacterial theory of the origin of the cysts feel that terramycin should be given in an effort to eradicate B. coli from the intestinal tract.[1] Several patients have undergone resection of most, or all, of the cyst-bearing intestines with relief of symptoms and without recurrence. In many instances the disease is too extensive for resection. The cysts have been noted to disappear spontaneously in many cases after the primary disorder, such as pyloric obstruction, has been corrected. Also, the cysts have been known to disappear without treatment in primary pneumatosis. Consequently, a period of watchful waiting is usually advised in the primary disease, followed by resection of the involved bowel if the disease is relatively localized, symptomatic and does not disappear spontaneously. Of course, intestinal obstruction may force surgical intervention. The possibility of a serious complication such as intestinal rupture must be borne in mind if the cysts do not disappear or increase in size.

SUMMARY

The first case of pneumatosis cystoides intestinalis complicated by a fatal rupture of the colon is reported.

The author is indebted to Dr. Chris Jackson, Danville, Kentucky, for clinical and pathologic data in this case.

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THE ROENTGENOLOGIC DETECTION OF SMALL RECTAL POLYPS*

A CONTRIBUTION TO THE PROPHYLAXIS OF CARCINOMA OF THE RECTUM

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SIXTY-FIVE per cent of carcinomas of the large intestine occur in the rectum.[2] The rectum is the site of 30 per cent of all gastrointestinal cancers. Approximately 500 patients die of cancer of the rectum each year in Massachusetts alone.[6] It is obvious that cancer of the rectum is a common and important disease.

Authoritative opinion is in agreement that cancer of the rectum usually has its origin in a simple, apparently innocuous, adenomatous polyp. Of the various methods available for the diagnosis of rectal tumors, roentgen examination is the one which is least used and most unreliable. The general accuracy in this country for the roentgen diagnosis of carcinoma of the rectum is said to be only 40 per cent.[1] Some[7] feel that to employ "a barium enema examination may be necessary to determine the condition of the bowel beyond the reach of the sigmoidoscope, or the degree of dilatation above an already detected rectal growth, but to employ it to detect such a growth itself is not only a waste of an expensive method, but is also dangerous, since it may not demonstrate the lesion." While digital and proctoscopic examinations are considered generally reliable and should always be employed in examination of the rectum, small lesions are, on occasion, overlooked. Proper importance must therefore be attached to roentgenologic examination because the roentgenologist is frequently the first consultant to examine a patient who complains of rectal bleeding or other rectal symptoms. An erroneous negative report may divert suspicion from this region and ultimately prove to be a fatal error.

Weber[8] was among the first to point out the difficulty in detecting polyps under 2 cm. in diameter and to stress the uncertainty which clouds the diagnosis. Later writers have concurred in his opinion.

The purpose of this paper is to show that even small rectal new growths can be found by careful roentgen examination. Since adenomatous polyps are precancerous lesions, their early detection and removal contribute to the prophylaxis of cancer at their sites.

It is well known that the large mass of barium introduced into the rectum by barium enema frequently obscures the lesion one is trying to find. This difficulty has largely been overcome by the development of a special technique which consists of spraying a thin mist of barium-water suspension on the rectal mucosa.[3] Routine roentgenologic procedures, however, have not been fully explored. We are definitely of the opinion that a straight lateral view, before and after evacuation of a barium enema, is an important contribution to the diagnosis of rectal and sigmoidal lesions (Fig. 1 and 2). Moreover, the lateral view frequently furnishes more information concerning the relationship of the rectum and sigmoid to their neighboring viscera than may be obtained by any other single diagnostic procedure.[4]

Examination by double contrast enema is also a necessary adjunct to the proper study of the colon for the detection of new growths. (In this connection, it may be of interest to state that for several years we have been using carbon dioxide instead of air. We have found it to be far superior to air with respect to safety to the patient and

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FIG. 1. Annular carcinoma of the rectosigmoid not visible on the posteroanterior roentgenogram (*A*) but clearly shown in the lateral view (*B*).

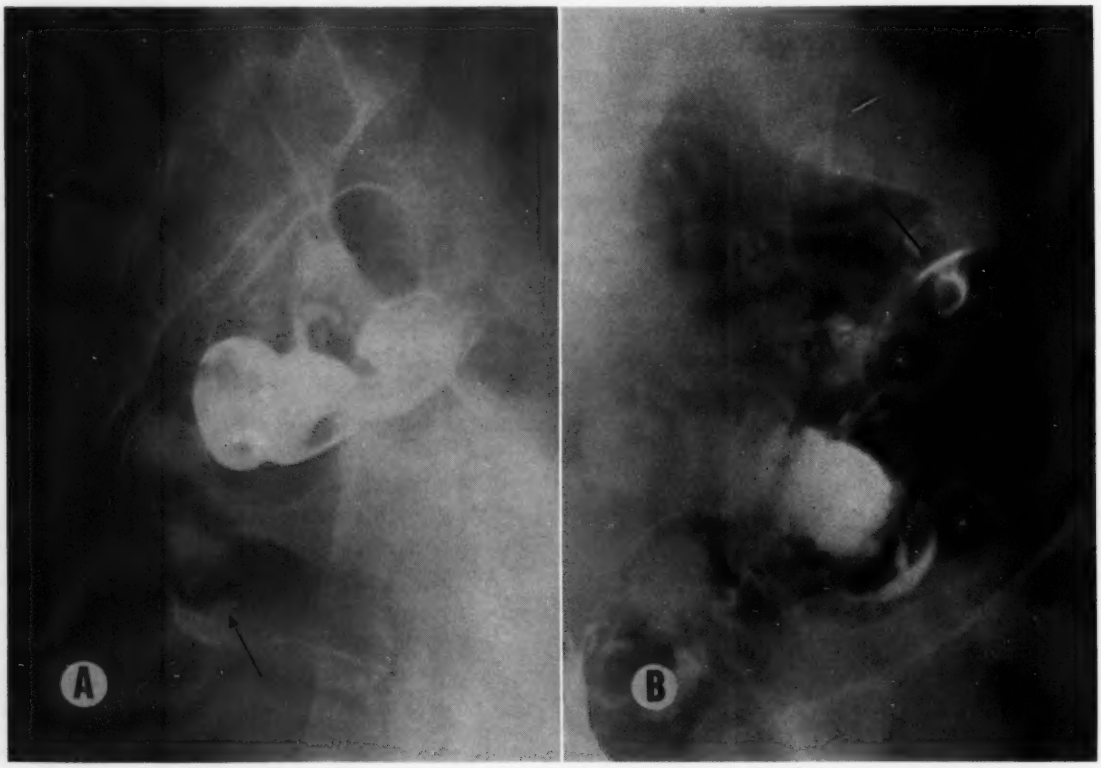

FIG. 2. Rectal polyp (arrow) not clearly visualized on the posteroanterior roentgenogram but well shown in in the lateral view (*B*). Confirmed by proctoscopic examination.

FIG. 3. Sessile polyp (arrow) of upper rectal ampulla as shown by spray technique. Note indentation of rectal margin. Confirmed by proctoscopic examination. Reprinted by permission of *Surgery*, 1949, *25*, 1, and the *American Journal of Digestive Diseases*, 1951, *18*, 6.

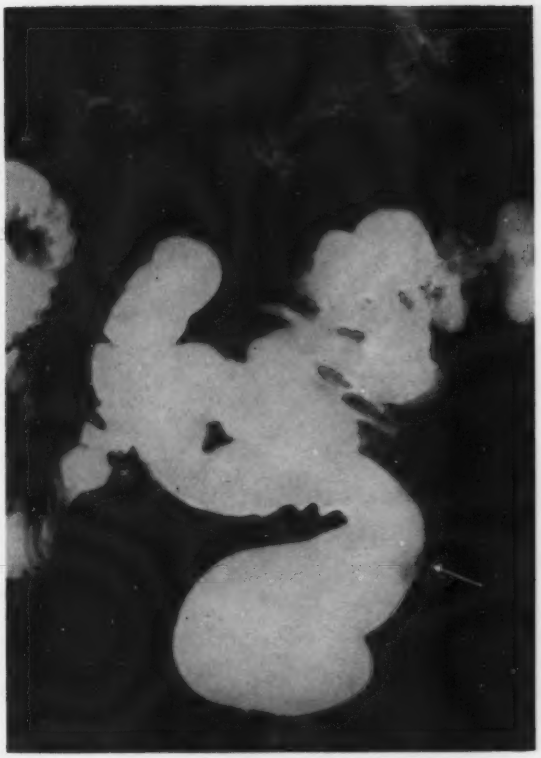

FIG. 5. Small polyp in lateral wall of the rectum (arrow). Note how the sharp normal border splits to encircle the polyp. Confirmed by proctoscopic examination.

FIG. 4. Polyp of upper rectal ampulla (arrow). Note the notched marginal defect. Confirmed by proctoscopic examination.

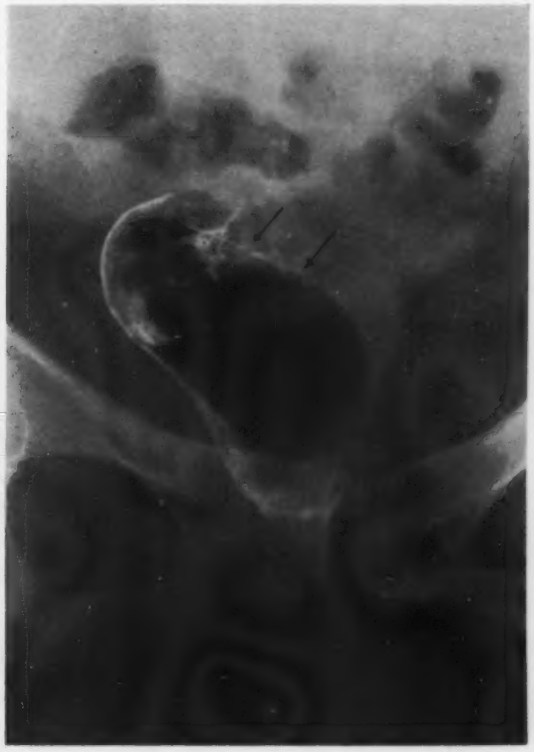

FIG. 6. Minute rectal polyps shown actual size (arrows). These measured 1 to 4 mm. in diameter. Note that the sharp rectal outline is interrupted. Confirmed by proctoscopic examination.

Fig. 7. (*A* and *B*) Small rectal polyp (arrow) shown actual size, visualized by double contrast enema. Confirmed by proctoscopic examination.

convenience for both patient and roentgenologist.[5])

The following illustrations (Fig. 3 to 9) are included with the hope that they may stimulate renewed roentgenologic interest in study of the rectum. It may be well to emphasize that, when distended, the normal rectal contour as seen in any view is bilaterally convex. Areas of flattening, indentation, notching or serration are abnormal and indicate the need for further study (Fig. 3 and 4). After evacuation, the normal rectal contour presents a smooth, sharply delineated contour. A break in this sharp line should arouse suspicion of a local new growth. Such defects may be only a few millimeters in size (Fig. 5 and 6), but, if constant, they are clinically significant. They are observed when the polyp is small and is located on the lateral rectal wall, or is seen tangentially. Larger polyps, or those centrally located, usually appear as ovoid or circular filling defects (Fig. 7, 8 and 9).

The cited examples of rectal polyps are typical of a large number which were found by roentgen examination. No claim is made that roentgenologic examination should take precedence over digital and proctoscopic examinations. But even on direct examination through a proctoscope, small polyps may be overlooked. Since the rectum must be filled in order to study any other portion of the large intestine, the roentgenologist is in position to discover unsuspected lesions of the rectum.

SUMMARY

The diagnostic accuracy of the roentgenologic examination for the detection of rectal new growths is far below the high standards achieved in the roentgenologic diagnosis of disease in other areas or systems of the body. Since cancer of the rectum is a common and serious disease, renewed efforts are called for in discovering the early pre-cancerous lesion. Examples

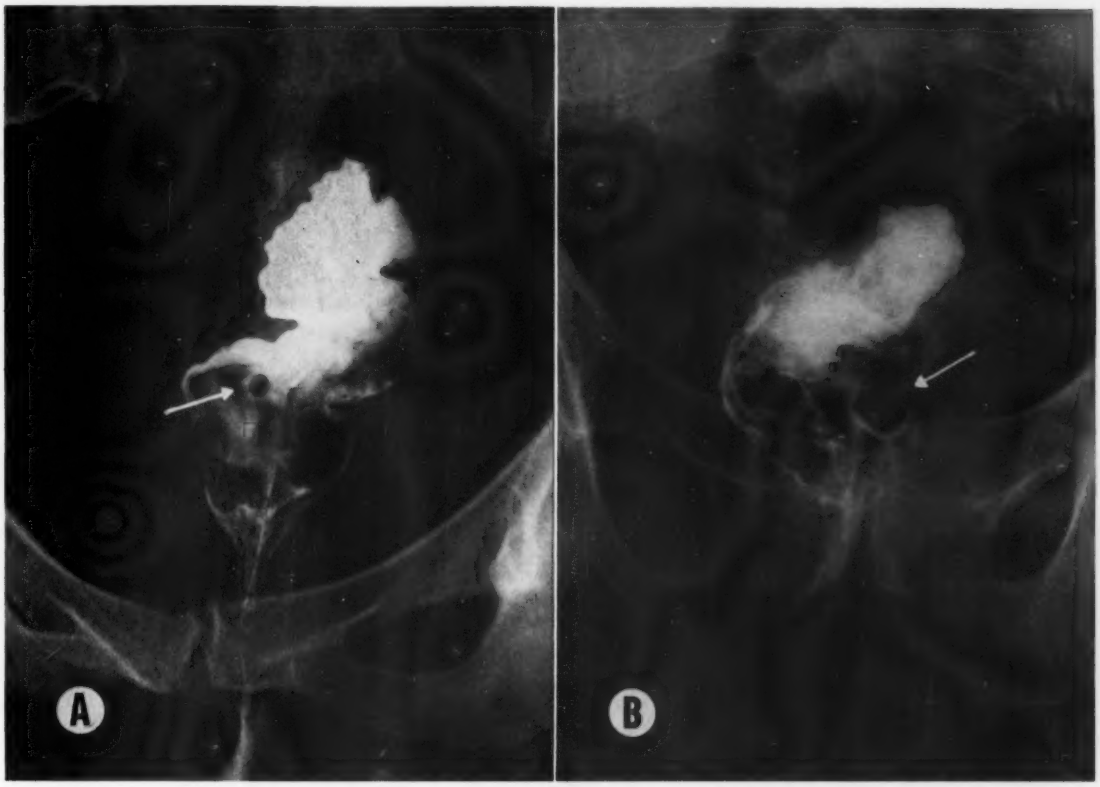

Fig. 8. (*A* and *B*) Rectal polyp (arrow) shown "en face" by spray technique. Confirmed by proctoscopic examination. Reprinted by permission of the *American Journal of Digestive Diseases*, 1951, *18*, 6.

are cited to show that even minute precancerous growths may be found by careful roentgenologic examination.

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Fig. 9. Rectal polyps 1 by 4 and 1 by 5 mm. (arrows) shown actual size. These were obscured by a plain barium enema, but were clearly shown by double contrast enema. The polyps were not seen on proctoscopic examination by an experienced and competent examiner. When they were pointed out to him on the roentgenogram, he was able to find and remove them.

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A VARIANT OF THE PSEUDOTUMOR SIGN*

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IN THE recent literature increasing attention has been devoted to the fact that fluid lying within closed loops of obstructed small intestine may be recognized roentgenologically. The shadow of the fluid-filled small bowel loop has been designated the "pseudotumor sign," and the finding was first described by Frimann-Dahl.[1–4] Other authors[5–7] have commented on this sign and have agreed that it usually is found in cases in which there is strangulation of the closed loop.

We have encountered an interesting variant of the pseudotumor sign in 2 patients. In both of these there was evidence of strangulation of the obstructed intestinal loop at surgery.

REPORT OF CASES

CASE 1. L.B., fifty year old Negro male, entered the hospital on October 16, 1951 with a five day history of acute midepigastric pain and vomiting. Vomiting had been episodic until the day of hospitalization when it became persistent, and neither food nor fluids taken by mouth could be retained. The only pertinent note in the past history was that the patient had undergone an abdominal operation twenty years prior to the current admission.

Physical examination revealed a man acutely and seriously ill. There was no evidence of abdominal distention or tenderness, and no masses were palpable. Occasional bowel sounds could be heard. On the day of admission roentgenograms of the abdomen (Fig. 1) showed the presence of air in the upper small bowel and two well-defined rounded areas of soft tissue density lying in the pelvis minor. These were clearly demarcated peripherally by thin radiolucent lines. It was suggested that these shadows represented fluid-filled loops of small intestine, but considerable controversy resulted in the administration of barium by mouth. Figure 2 shows opacification of the stomach and upper small bowel by barium, and graphically shows barium opacification of a fluid-filled loop in the pelvis. The persistence of the dilated loop in the same position which it occupied on the previous day was evidence that it was fixed. A diagnosis of obstruction of the ileum on the basis of old adhesions was made and surgery was performed.

At the operating table, after much of the retained fluid and barium had been removed by means of suction through a Miller-Abbott tube,

FIG. 1. Case 1. Anteroposterior roentgenogram of the abdomen shows air in small amounts in upper small bowel and in transverse colon. The principal finding of interest is the presence of two well-defined cyst-like areas of soft tissue density with polycyclic borders which lie just above the shadow of the bladder in the pelvis minor.

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Fig. 2. Case I. Anteroposterior roentgenogram of the abdomen made after the ingestion of barium mixture shows opacification of dilated and fluid-filled loops of small bowel. The double cyst-like shadows in the pelvis are identified as a loop of small bowel seen on end. The point of obstruction lay in the distal aspect of this loop. This figure represents an examination performed twenty-four hours after that seen in Fig. 1.

an obstruction low in the ileum caused by adhesions between loops was identified and relieved. Although the loops were immobilized, the involved bowel was completely vital.

Case II. H.S., a seventy-nine year old white male, was admitted to the University Hospitals on March 21, 1953 with a two day history of lower abdominal cramps. Early in the present illness the pain had seemed to radiate to the epigastrium. The patient complained of nausea and many bouts of emesis. Some of the vomited material was said to have contained blood. An enema had been productive of a few black fecal masses. The patient was known to be suffering from peripheral arterial insufficiency, and past history revealed that he had had suprapubic prostatectomy and appendectomy. Physical examination showed the abdomen to be slightly distended, and bowel sounds were audible.

On admission, anteroposterior recumbent and erect roentgenograms of the abdomen showed air in loops of small bowel in the mid-abdomen. Air was seen in several dilated loops of small bowel lying vertically in the upper abdomen and above the bladder shadow three fluid-filled and dilated loops of small bowel were identified (Fig. 3 and 4). A diagnosis of low small bowel obstruction of a strangulating nature was made on the basis of the fluid-filled loops. Once again the pseudotumors seen in the pelvis minor were interpreted as fluid-filled loops of ileum visualized on end.

At surgery there was evidence of volvulus of the distal ileum about old adhesions near the site of the previous appendectomy. The involved bowel was tensely distended by fluid and was clinically gangrenous.

Fig. 3. Case II. Anteroposterior roentgenogram of the abdomen shows the presence of air in small bowel loops in the upper abdomen, some of which appear to be dilated. Three cystic shadows with somewhat radiolucent margins are identified in the pelvis minor.

Fig. 4. Case II. Erect anteroposterior roentgenogram of the abdomen which shows very little fluid in the gas-filled small bowel loops, but shows no change in position of the three soft tissue densities in the pelvis minor. These loops were identified surgically as fixed, fluid-filled intestine at the site of obstruction.

DISCUSSION

Two cases have been presented in which a pseudotumor sign was present in the pelvis, represented by round masses of soft tissue density with radiolucent margins. The lucent shadows might well be thought to represent subserosal fat, a goodly amount of which was present in each of the 2 patients. In each of the cases the findings were shown to be due to the presence of a closed and fluid-filled loop of small bowel lying in the pelvis minor which was visualized on end, lying parallel to the roentgen beam. In 1 case the nature of the pseudotumor shadow was demonstrated by preoperative barium examination.

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THE USE OF A WATER SOLUBLE CONTRAST MEDIUM (HYPAQUE) FOR GASTRO-INTESTINAL ROENTGENOGRAPHY*

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WATER soluble contrast media offer certain advantages in gastrointestinal roentgenography which merit further study. Occasionally, a gastrointestinal examination is required under circumstances in which the use of barium sulphate suspensions is questioned as, for example, in the presence of possible perforations, hemorrhage or certain obstructions. The use of 30 per cent and 70 per cent urokon was reported by Canada,[1] who found that these substances had an extremely bitter taste which made necessary their introduction into the stomach by means of a tube. Although the stronger solution had been used by her in 2 patients with no adverse effects, the weaker concentration was employed more often. Studies of fistulas leading into abscess cavities showed rapid disappearance of the medium, presumably by absorption or diffusion. Some absorption from the gastrointestinal tract was indicated by faint or distinct pyelograms a short time after the introduction of urokon into the stomach. Allergic reactions or iodinism were considered theoretic possibilities. A poor coating effect on the lining of the stomach was noted. Advantages of urokon and hypaque in gastrointestinal roentgenography were reported by Davis, Huang and Pirkey,[2] who also instilled the material into the stomach through gastric tubes. A slight diarrhea appeared in their patients, attributed to the osmotic activity of the material. Moore[4] suggested that diiodone be swallowed or injected into the stomach by means of a tube as an aid to the identification of perforated ulcers.

Recently, we were supplied with a 90 per cent solution of hypaque intended for angiocardiography. This substance is an aqueous mixture of 0.3 gm. of the sodium salt and 0.6 gm. of the methylglucaine salt of 3,5-diacetamido-2,4,6-triiodobenzoic acid per cubic centimeter. Each cubic centimeter supplies 0.462 gm. of iodine. At room temperature the material appears as a suspension of crystals, but is readily brought into a clear solution at body temperature. The commercially available 50 per cent solution of hypaque was similarly used. Later we were also supplied with hypaque in powder form. By eliminating the buffering, sterilization and ampule

FIG. 1. Sinus tract visualization after the injection of 90 per cent hypaque solution. The stomach and duodenojejunal junction are visualized. A large jejunal diverticulum is visualized.

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packaging, the cost of this material should be less than that supplied for angiocardiography. It can be made up to desired strength by diluting with water, and can be flavored with various agents to disguise its taste. We have used vanilla extract, oil of orange and sorbitol.

The first opportunity to use the 90 per cent hypaque solution was in a patient with a fistula in the right upper quadrant which appeared after subtotal gastrectomy for duodenal ulcer. The fistula was well outlined after injecting the hypaque into the opening on the skin and could be seen entering the duodenojejunal area and the stomach. There were no untoward reactions.

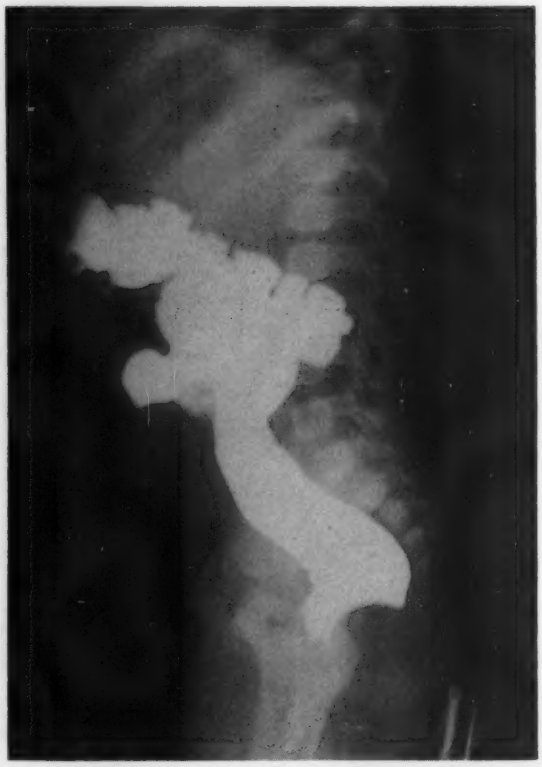
The study was continued by giving adult patients 90 per cent hypaque. Quantities varying from 25 cc. to 150 cc. were given to 20 unselected patients and

Fig. 2. Roentgenogram made one hour after the ingestion of 75 cc. of hypaque solution. Note that while the stomach still contains a large amount of the contrast medium, the lesser bowel and colon are well visualized.

Fig. 3. Roentgenogram made after the ingestion of 25 cc. of 50 per cent hypaque solution in a thin patient who had had a recent subtotal gastrectomy for duodenal ulcer.

opacification often comparable to that of barium was produced. The rugal folds of the stomach, the duodenal cap and sweep, and the jejunal loops could be studied adequately. The passage of this medium through the small intestine was rapid, so that within one to two hours the entire lesser bowel had been traversed and the hypaque could be seen in the colon. Good mucosal patterns of the lesser bowel were not obtained, the various loops being moderately dilated. The transit time through the large intestine was such that opacities were seen in the colon after twenty-four to forty-eight hours.

The 50 per cent hypaque solution was used in a similar fashion in 10 cases, and adequate opacification was obtained in children and thin adults. One patient had had a cholecystogastrostomy and gastroenterostomy for carcinoma of the head of the pancreas. A scout roentgeno-



gram revealed air in the hepatobiliary radicles and the gallbladder. A gastrointestinal examination with 50 per cent hypaque outlined these structures with no untoward effects. Serial studies disclosed that after two hours a precipitate of almost metallic density appeared in the stomach which moved freely and sedimented with the patient in the erect posture. This was attributed to the fact that hypaque may flocculate in a high acid medium.

The only significant untoward effect in this group of patients was a tendency to mild diarrhea. This appeared in patients given the dilute as well as the concentrated solution. From two to six watery stools were observed in 4 patients within eight hours. There were no complaints of nausea, vomiting or pain. One patient with known

Fig. 4. Lateral roentgenogram of the colon after enema using 25 per cent hypaque solution in an eighteen month old infant.

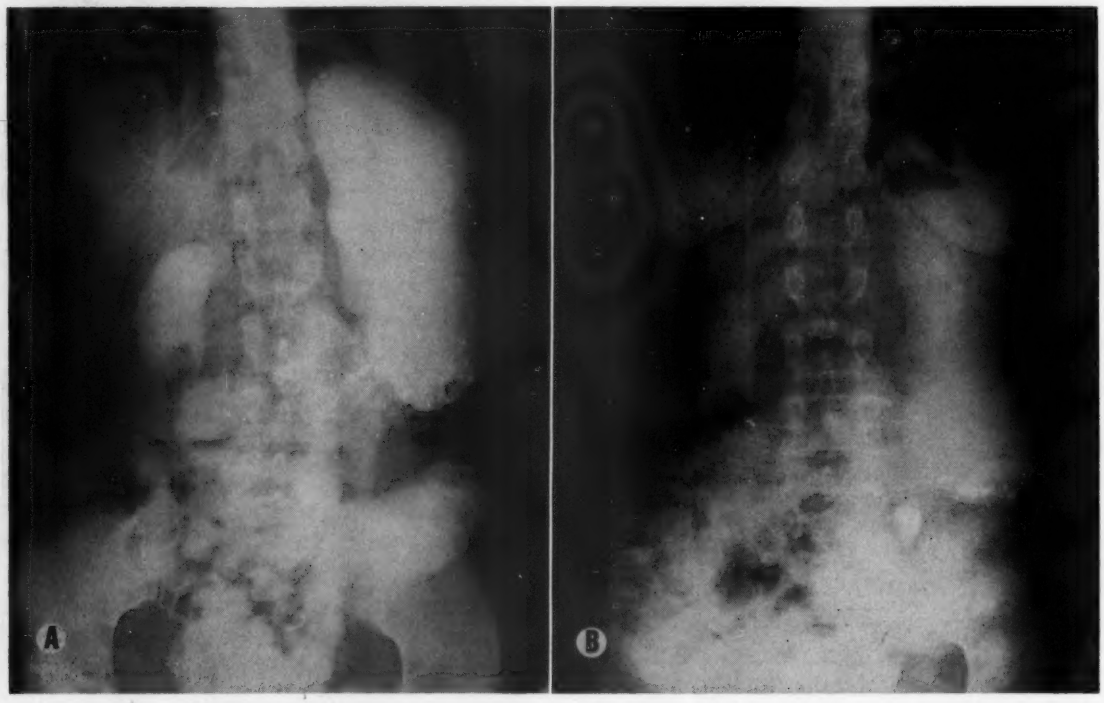


Fig. 5. (*A*) Supine roentgenogram made two hours after the ingestion of 50 cc. of 50 per cent hypaque solution. The contrast medium has entered the gallbladder and biliary radicals through a cholecystogastrostomy. Note the opaque medium along the greater curvature of the stomach. (*B*) Same patient, erect study. The densities in the stomach have formed a layer.

allergies had giant hives which disappeared after an antihistaminic was given by mouth. No visualizations of the urinary tract occurred in this series.

Hypaque solution was found useful in colon examinations, particularly in children. In infants and young children the material may be diluted to 25 per cent or even less. Postevacuation roentgenograms showed good emptying of the bowel, but the mucous membrane patterns were poorly demonstrated. One case of more than ordinary interest was a child who developed recurrent intussusception following operation in whom hydrostatic reduction was accomplished with hypaque solution.

A vexing problem encountered in daily practice is the differentiation of physiologic ileus from mechanical obstruction. The interpretation of scout roentgenograms is open to much discussion and difference in opinion. The administration of barium mixtures to such individuals is generally considered inadvisable even though competent authorities[3] advocate its use. We have attempted to compromise this issue by giving hypaque solution in such cases. The additional opacification of the lesser bowel contents and the presence of hypaque in the colon within a few hours has proved helpful in several instances, and further work along these lines is in progress.

SUMMARY

Hypaque solutions are useful in the investigation of the gastrointestinal tract and for sinus tract visualizations. The unpleasant taste may be masked with flavoring agents. Aside from diarrhea, very little untoward effect is noted. The urinary tract was not visualized after the oral ingestion of this medium.

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DETECTION OF GASTROINTESTINAL BLEEDING WITH A BARIUM-HYDROGEN PEROXIDE MIXTURE*

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THE detection of various pathologic processes in the upper gastrointestinal tract and small bowel by the usual barium gastrointestinal series is most rewarding. There remains a number of individuals with gastrointestinal bleeding who have no demonstrable cause. Some of these patients have only superficial examinations due to their serious condition, or for fear of "stirring things up." Others may be carefully examined in detail and yet the answer is not apparent. How delightful it would be to have a method of discerning the level or location of the bleeding. One's attention could then be turned to this point, and the diagnosis would probably yield to close scrutiny.

Most laboratory tests for the detection of blood revolve about colorimetric reactions. These are of little help in this specific problem. To be of roentgenographic help, a medium with changing physical properties of roentgenographic density or character when in contact with blood is required. If the barium could be precipitated or clumped, it would be visible; however, this does not seem to be feasible. A change in density and character of the barium would be equally acceptable. This can be accomplished by the addition of hydrogen peroxide, which releases oxygen and foams on contact with blood. The result *in vitro* was an easily discernible pattern of tiny bubbles throughout the barium. Two to three teaspoonsful (8 to 12 cc.) of hydrogen peroxide in one 6 ounce cup of Veriopake was quite sufficient. If two or three drops of blood were added and the mixture agitated, small bubbles occurred throughout (Fig. 1). Further agitation actually gave a"head" of foam. The stirring in of more blood exaggerated the process, but many of the bubbles coalesced and left the suspension. A few drops of blood were seen almost as easily as when several cubic centimeters were used.

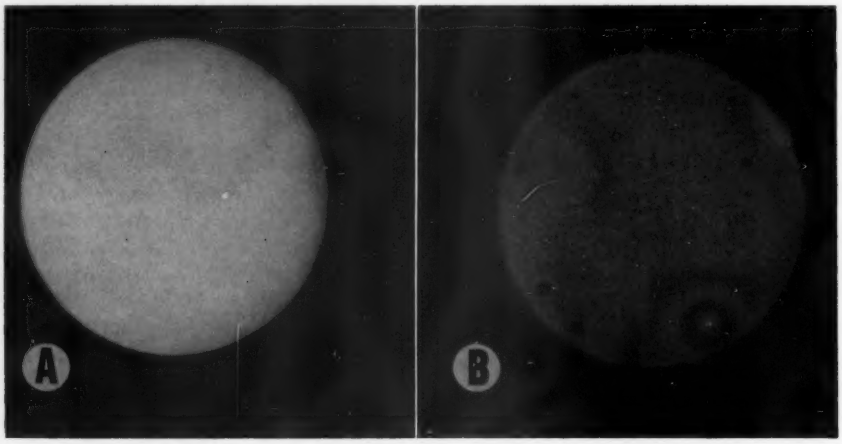

FIG. 1. (*A*) Roentgenogram of a paper cup containing Veriopake and hydrogen peroxide. (*B*) The same as *A* plus a few drops of blood and agitation. Innumerable tiny bubbles are dispersed throughout. The larger bubbles are foam on the surface.

* From the Department of Radiology, The Medical Group, Honolulu, Hawaii. Presented at the Annual Meeting of the Hawaii Medical Association, May, 1955.

Hydrogen peroxide as an oxidizing agent is broken down to water and oxygen by reducing agents and certain organic compounds, including blood and pus. When the barium-hydrogen peroxide medium was ingested, some slight foaming occurred in the mouth, but not sufficient to be visible roentgenographically. The same was true of the stomach and small bowel contents. Saliva or aspirated gastric secretions from normal individuals showed no foaming *in vitro*. The morning gastric lavage from a pulmonary tuberculous patient was similarly negative. Fecal matter, however, contains agents which invalidate its use in the colon for this purpose. In the fasting person the breakdown of hydrogen peroxide and resultant foaming in the esophagus, stomach or small bowel was essentially specific for blood. Hydrogen peroxide is safe and cannot affect the body as a whole. It is impossible for it to exist in the blood stream. Locally it is a mild styptic and promotes the coagulation of blood.[2,3] The taste varied when described by different individuals, but it was generally disliked, and in the manner used here occasionally caused mild nausea.

Preliminary experiments *in vivo* were rather disappointing. The ingestion of blood by an individual, followed by the barium-hydrogen peroxide, gave a roentgenographic image that was smudged. The barium simulated the appearance seen when the stomach contained a large amount of residual food or secretion. The tiny bubbles, so well seen *in vitro*, were not apparent, presumably due to the scattered radiation and loss of fine detail. This smudged appearance of the barium was undoubtedly produced by the bubble formation interspersed throughout the medium. It did not differ sufficiently from that seen with other causes to be of certain value alone. Larger bubbles or foam were apparent in the erect position or on the air contrast views. This could be seen with ease in some, while in others it required multiple roentgenograms. These large bubbles and the appropriate views for their demonstration were required to indicate the presence of blood *in vivo*. It was also apparent that one drop of blood was not sufficient to be seen. One cubic centimeter or more would give good results.

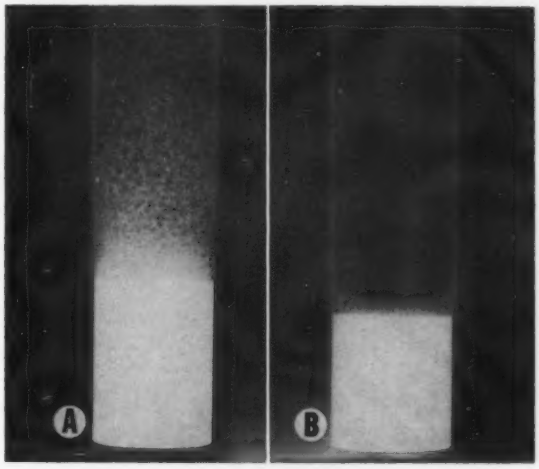

Fig. 2. (*A*) Veriopake, hydrogen peroxide, and blood mixture showing foam remaining 80 minutes after agitation. (*B*) Micro-barium, hydrogen peroxide, and blood mixture at the same time. Very little foam developed and little persisted.

The tiny bubbles seen *in vitro* were apparent with any hydrogen peroxide-barium suspension. The foam was impressive only when Veriopake and hydrogen peroxide was used. With plain barium sulfate or with a micro-barium sulfate suspension, the foam was difficult to produce and remained for only a fleeting moment. The pectin and tartaric acid in the Veriopake evidently stabilized the foamy bubbles. Figure 2 shows a roentgenogram taken 80 minutes after mixing blood in a tube with hydrogen peroxide-Veriopake and a second tube of hydrogen peroxide-micro-barium suspension. The foam in the latter was minimal and disappeared immediately. The foam with the Veriopake mixture was excellent and remained almost the same 24 hours later. It was dispersed by further shaking.

Since the inception of this experiment in 1953, Klami[1] has published the results of a similar procedure on 40 cases in an attempt to differentiate carcinoma and benign lesions. This proved to be helpful in the case of esophageal carcinoma and stricture;

Table I

	Cases without Bubbles	Cases with Bubbles	Total
Negative (including recheck healed ulcers, hyperperistalsis, pylorospasm, subtotal resections, etc.)	49	2	51
Hiatal hernia	8	1	9
Gastric ulcer, benign	7	5	12
Gastric polyp	1	1	2
Stomal ulcer	2	0	2
Pyloric stenosis, adult	1	0	1
Gastric carcinoma	2	0	2
Hypertrophic gastritis	9	1	10
Ulcerative gastritis	0	1	1
Duodenal ulcer, active	36	6	42
Duodenal ulcer scar without crater	8	0	8
Duodenitis	6	0	6
Anomalies	2	0	2
Esophagitis	1	0	1
Carcinoma of the pancreas	1	0	1
Total	133	17	150

foam appeared with the former, while the strictures showed none. In the stomach most carcinomas produced foam, but not always. Gastritis hemorrhagica produced the larger amounts of foam and did so consistently.

A hydrogen peroxide-Veriopake mixture was used by us in 150 consecutive gastrointestinal examinations. Table 1 summarizes the conditions found and lists whether bubble formation and presumably blood were demonstrable. In the two negative cases these bubbles were quite small, but nevertheless visible. Bubble formation was seen in over 40 per cent of the cases with gastric ulcer, but in only 1 out of 7 cases of duodenal ulcer (Fig. 3, 4 and 5). Several cases had a past history of tarry stools, but none within five days before the examination except for the case of the hiatal hernia with bubble formation. This patient had several recent tarry stools, and the obvious bubble formation in the herniated stomach pouch gave evidence that a localized gastritis must be accounting for the bleeding. One individual showed bubble formation in the duodenal cap and no evidence of ulcer. This prompted a re-examination the same morning after the two hour serial film, at which time a duodenal ulcer was clearly demonstrated. At the first study the crater was apparently filled with

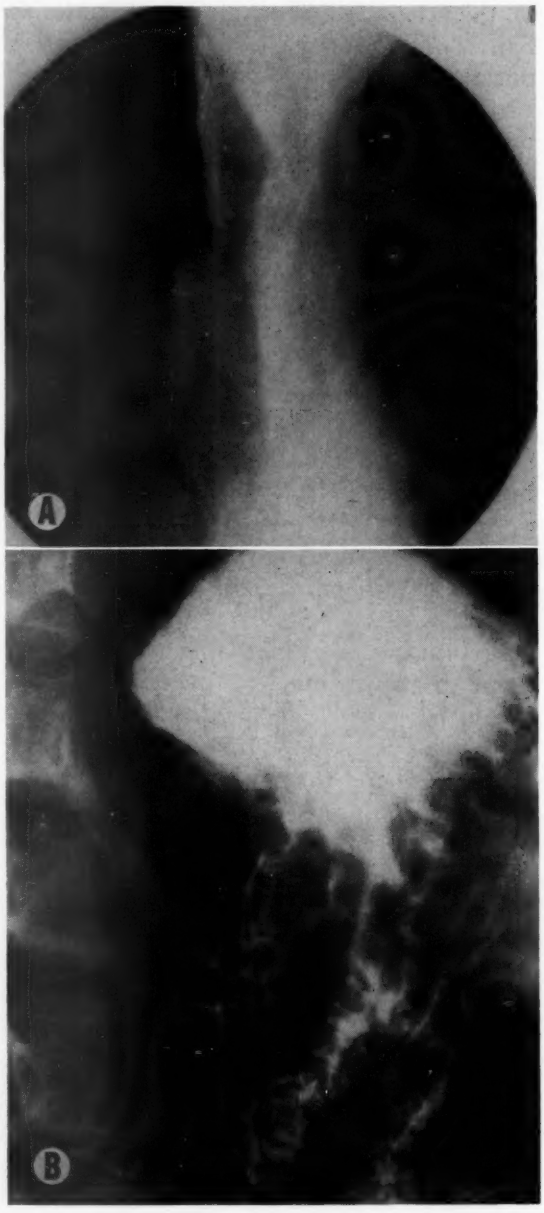

Fig. 3. (*A*) Pressure "spot film" of benign lesser curvature gastric ulcer. (*B*) Air contrast view demonstrating foam formation just below the ulcer crater indicating the presence of blood.

Fig. 4. Benign prepyloric gastric ulcer and duodenal ulcer (obscured on this view) showing bubble formation in the antrum and duodenal cap due to the presence of blood.

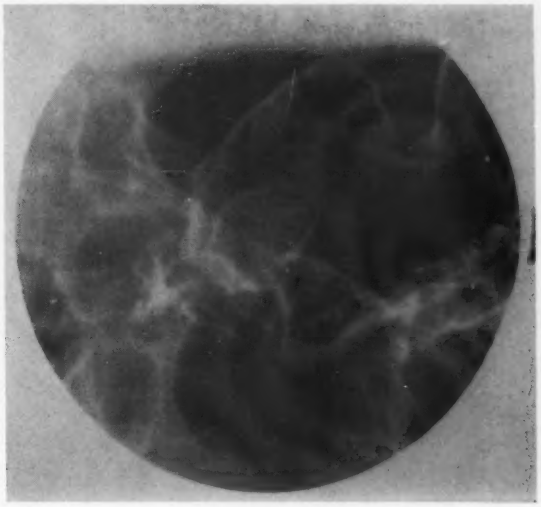

Fig. 6. Bubble formation seen in the antrum of the stomach on the air contrast view. A large ulcerating carcinoma involved the gastric body and fundus.

debris. The 2 cases with carcinoma of the stomach and 1 other recently examined showed a very smudged appearance of the barium, suggesting the presence of tiny dispersed bubbles. No foam could be demonstrated in these first 2, but gross bubbles were present in the third (Fig. 6). All 3 were of the ulcerative type and with guaiac positive stools. The lack of bubbles in the first 2 was difficult to explain with certainty. At that moment there may have been little or no blood. More probably it was the result of neoplastic limitation of peristalsis and resultant poor mixing. Agitation was required to produce the large bubbles *in vitro*. Regardless of the cause, the medium was of no help in differentiating between gastric benign and malignant lesions. The

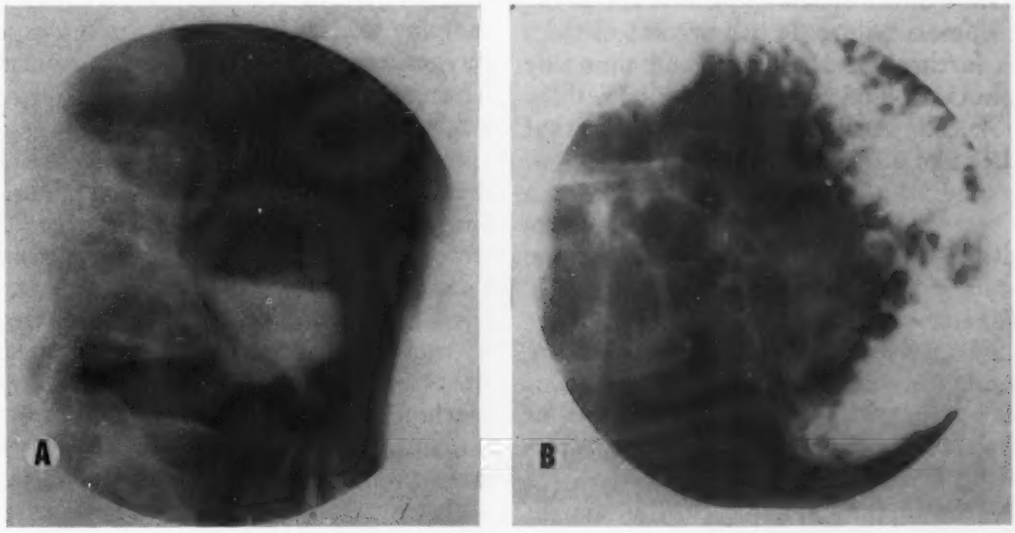

Fig. 5. Two cases of active duodenal ulcer with bubble formation and presumed blood. (*A*) Erect position to show the foam floating above the barium. (*B*) Air contrast view of the duodenal cap to show the bubbles. The ulcer craters are obscured here, but were easily seen on routine projections.

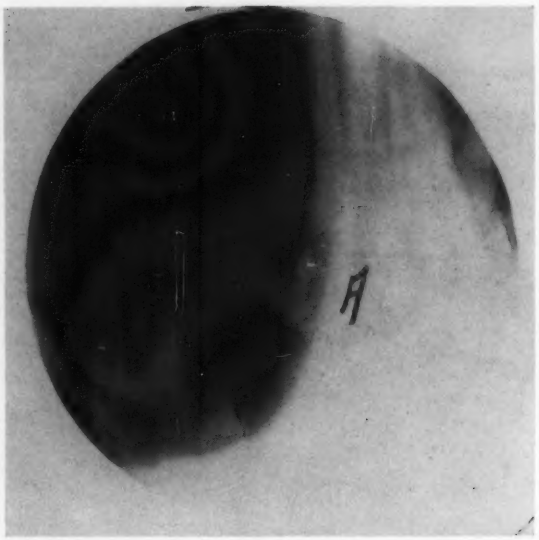

Fig. 7. Ulcerative gastritis with multiple shallow ulcers on the lesser curvature at A, and bubbles accumulated in the antrum (arrow).

one case of superficial ulcerative gastritis produced profuse bubble formation (Fig. 7).

All the cases showing bubble formation had guaiac positive stools, except for the 2 negative ones mentioned above. Three cases of duodenal ulcer and 1 of gastric ulcer with guaiac positive stools showed no foaming. The failure to show evidence of blood in these latter cases was due to the cessation of the bleeding, insufficient blood, or technical failure. It has become obvious with further use of this method that tiny amounts of blood will not always be demonstrable. The sensitivity is not equal to that of the stool guaiac test. It requires blood at the time of the examination, at least a cubic centimeter or more, on the basis of ingested trials. The differential from normal foaming is discussed below.

The demonstration of bubbles at one site naturally does not indicate that the bleeding originated there. It points out that bleeding occurred at this point or above. In all of the cases of duodenal ulcer showing bleeding by this method, bubbles were seen in the duodenal cap but not in the stomach. In the case of the gastric ulcers, bubbles were usually visible at or near the ulcer site with the air contrast views and occasionally in the duodenal cap, even when no crater was present at the latter point. The bubbles seemingly formed there as a result of the blood having been carried down from above, rather than the bubbles being propelled along.

Films of previous gastrointestinal studies were then reviewed to assay the possibility of bubble formation when a Veriopake medium without hydrogen peroxide was used. Much to my surprise, an occasional film demonstrated foam. It was soon obvious that bubbles in the esophagus were commonly seen in the normal individual with or without hydrogen peroxide medium. In the stomach and duodenal cap, bubbles without hydrogen peroxide were never impressive. After the first swallow of barium, a mucosal pattern film would show some few bubbles. In the cases having an appreciable gastric residue, bubble formation occasionally appeared in the duodenal cap. The bubbles were never too impressive, and their temporary nature differed sufficiently to lead to only moderate confusion. Their occasional presence without hydrogen peroxide medium is nevertheless quite real and this detracts from the certainty of the method. On the other hand, the smudged appearance of the barium produced by the multitude of tiny bubbles plus foaming was seen only in those cases with obvious evidence of bleeding by stool test. The combination seemed to give positive evidence of bleeding.

Since bleeding from the stomach and duodenum obviously placed blood in the small bowel, bubbles would have been expected with the hydrogen peroxide-barium medium in this location. In practice these were not impressive. Presumably the hydrogen peroxide or the catalytic effect of the blood exhausted each other before reaching this area. A loop of jejunum would occasionally contain a few bubbles, which at first was thought to indicate blood. After reviewing the films of non-hydrogen peroxide medium, some loops of small bowel were seen to contain bubbles to an equal degree. This was particularly evident in

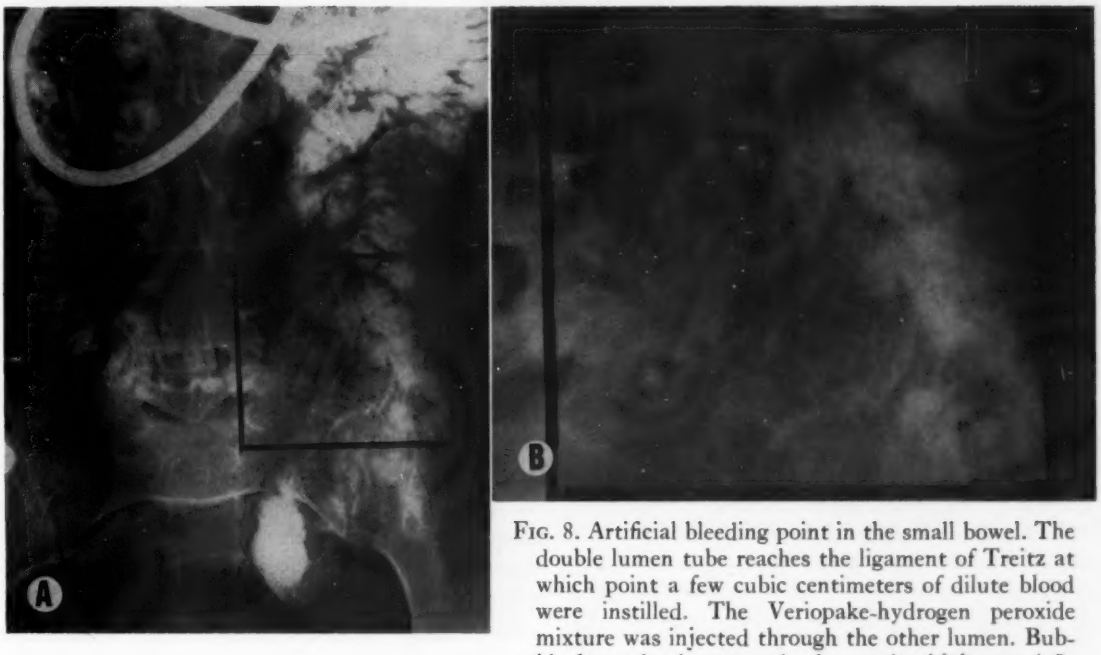

Fig. 8. Artificial bleeding point in the small bowel. The double lumen tube reaches the ligament of Treitz at which point a few cubic centimeters of dilute blood were instilled. The Veriopake-hydrogen peroxide mixture was injected through the other lumen. Bubble formation is present in the proximal jejunum (*A*), but best seen in the mid-jejunum (*B*) after adequate mixing occurred.

those patients having a short transit time through the small bowel. The hyperperistalsis thrashed the barium mixture about enough to produce some foam. Fortunately this moderate bubble formation was quite transitory as roentgenograms taken a few moments later showed the bubbles to have disappeared. Since bubble formation occurs in the normal small intestine, it detracted from the accuracy and value of the hydrogen-peroxide medium to detect bleeding originating in this region. Nevertheless, this method proved to be of value in this location, since when both foam and a smudged appearance occurred together, the findings have been correct. The bleeding nonobstructing small bowel neoplasm or ulceration is difficult to detect. Often these lesions escape even the most careful examination. If they are bleeding, the hydrogen peroxide-barium mixture may point to their location. No case of this nature appeared in the present series, and conclusions cannot be formed on this most interesting facet other than those below.

A bleeding small bowel lesion was simulated by passing a double lumen tube into the fourth part of the duodenum of a normal individual. A Veriopake-hydrogen peroxide mixture was instilled through one lumen of the tube and a little blood diluted with water through the other. Obvious foam was demonstrable in the proximal and mid-jejunum with a smudged appearance to the barium (Fig. 8). At no time was bubble formation apparent at the tip of the tube, the site of the artificial bleeding point. Not until the medium had been propelled on for another loop or two were the bubbles visible. Agitation or proper mixing was required, as was to be expected from the *in vitro* tests. Objective examination of the roentgenograms in this artificial case was not so distressing as the above description may sound. It was quite clear that the bleeding point must be in one of three loops of small bowel. Although the exact point was not elucidated, it narrowed the region down to this short segment of the distal duodenum or very proximal jejunum.

DISADVANTAGES

1. Most individuals do not relish the barium suspension mixtures used for rou-

tine gastrointestinal examinations. The addition of hydrogen peroxide promoted an increased number of complaints about the unpleasant taste and palatability. This feature was not too distressing. An occasional patient will develop a mild epigastric burning and at times nausea. There was no vomiting and no patient refused to take the medium.

2. Due to the bubble formation in the presence of blood in the duodenal cap, difficulties were occasionally experienced in obtaining satisfactory erect pressure films of this area. The continued release of free oxygen produced sufficient gas to give a fluid level, even when the cap was earlier free of gas. This interference could be overcome by the usual procedure of forcing more barium through with finger pressure or temporarily placing the patient in the prone position with the left hip elevated. It did not interfere with the prone films.

3. Bubbles but no smudging were occasionally seen without hydrogen peroxide in the normal individual, rarely in the stomach or duodenum but not too infrequently in the small intestine. This detracted from the accuracy. It particularly required further time, attention, and additional roentgenograms by the examiner to make a differentiation between these smaller temporary bubbles and the foam produced by blood.

ANTICIPATED VALUE AND USE

The maneuvers required to demonstrate foam make the upper gastrointestinal examination more complicated and time consuming. Because of this and the other above disadvantages, the use of this mixture is now limited to those patients having a history of bleeding, either "tarry stools," or fecal occult blood. Here the value seems real and for the last year and a half it has proved itself helpful in those cases where the bleeding abnormality was not otherwise apparent.

SUMMARY

A hydrogen peroxide-Veriopake mixture will bubble and foam when mixed with blood. This foam and a smudged appearance of the barium were demonstrable roentgenographically *in vivo* and pointed to the general location of the bleeding lesion. The method proved valuable in those cases having a current or recent history of gastrointestinal bleeding. Air contrast and erect views of the stomach and duodenal cap were needed to demonstrate the foam in these locations. The bubble formation was not absolutely specific, as some temporary foam does occasionally occur in the normal individual.

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THE USE OF RADIOACTIVE ISOTOPES IN THE EVALUATION OF GASTROINTESTINAL FUNCTION*

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THE need for an effective and simple test to measure the uptake of fat from the gastrointestinal tract has long been recognized. Radioisotope tracer techniques lend themselves ideally to this type of study. During the past two years we have developed and applied a test utilizing a radioactive tagged fat meal.[1]

TECHNIQUE

The fat test meal consists of an emulsion containing 48 per cent peanut oil, 48 per cent water, 4 per cent tween 80 (an emulsifying agent), and 25 microcuries of I^{131} labeled glycerol trioleate (0.05 to 0.1 ml.). One milliliter of this emulsion is given per kilogram of body weight. Twenty grams of barium sulfate are mixed with the test meal so that its progress through the gastrointestinal tract can be followed roentgenographically. Twenty drops of Lugol's solution are given immediately following the test meal to effectively block the thyroid uptake of radioiodine. The patients fast for six hours prior to the ingestion of the test meal and for the following six hours, after which normal eating habits are resumed.

Blood samples (2 ml.) are drawn at the four, five and six-hour periods after the test meal. Gastric emptying is determined by roentgenographic examination.

A scintillation well counter is used for the radioanalysis of the individual blood samples. Using the measured radioactivity of the 2 ml. sample and the calculated blood volume of the patient, the total radioactive content of the blood is calculated and expressed as a percentage of total ingested radioiodine.

All feces are collected, urine free, for forty-eight hours following the test meal. Fecal samples are collected in individual containers which are sealed and sent to the laboratory for analysis. Each sealed container is analyzed for radioactive content and the total is expressed as a percentage of the test meal.

This test, when applied to 33 normal humans and 25 dogs, gave characteristic and reproducible results. The amount of radioactivity measured in the blood reached a plateau between three and four hours and held in this vicinity for the remainder of the six-hour period. The average level of this plateau was between 12 and 13 per cent of the ingested material.

The average fecal recovery for the 33 human controls was 0.6 per cent with a maximum of 1.4 per cent.

The effect of gastric emptying on blood levels was investigated. In dogs, when the pylorus was occluded, there was no significant absorption of radioiodine. In humans, delay of gastric emptying resulted in initially low blood levels.

Pancreatotomized dogs were also studied and blood level plateaus were found to be depressed to a level of 2.0 to 2.5 per cent as compared to the normal of 12 to 13 per cent.

Bile duct occlusion in a group of dogs showed minor changes in the blood level plateau, indicating that bile plays a minor role and pancreatic juice a major role in fat digestion and absorption.[6]

CLINICAL APPLICATION

The test was applied to a group of 31 patients with verified pancreatic disease.[5] Nineteen of the group had carcinoma of the

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Table I

RESULTS OF FECAL STUDIES

	Number	Abnormal	Abnormal	Average Fecal Recovery Per Cent	Range Per Cent
Normal	25			0.6	0 to 1.4
Functional diarrhea	44	0	0	1.2	0 to 2.8
Chronic pancreatitis	8	7	87.5	31.2	0.4 to 61.0
Carcinoma of pancreas	11	9	81.3	34.5	1.9 to 93.7
Hofmeister resection	48	26	54.0	13.8	0 to 57.4
Vagotomy and gastroenterostomy	18	11	61.0	18.0	0.4 to 52.3
Billroth 1 resection	8	3	37.5	4.5	0.2 to 8.6
Whipple's disease	1	1	100	60.5	
Sprue	1	1	100	66.5	
Regional enteritis	3	3	100	57.4	33.4 to 77.4

pancrease, 12 had chronic pancreatitis. Eighteen of the 19 cases of carcinoma gave abnormal tests as evidenced by low blood levels and high fecal determinations. Ten of the 12 cases of chronic pancreatitis showed low blood levels and high fecal levels (Table I and II).

A large group of postoperative patients was studied to evaluate the effects of the procedure upon fat digestion and absorption.[3] A total of 83 patients of this type were investigated and grouped as follows: Partial gastrectomy (Hofmeister) 48, partial gastrectomy (Billroth 1) 12, and vagotomy with gastroenterostomy 23. Of those having the Hofmeister procedure, 47 per cent had abnormal blood levels and 54 per cent had high fecal levels. In the Billroth 1 group, 42 per cent had abnormal blood levels and 37.5 per cent had abnormal fecal fat recovery. In the vagotomy with gastroenterostomy group, 73 per cent had abnormal blood levels and 61 per cent had high fecal fat (Table I and II).

Six patients with small bowel disorders were studied and all showed abnormal tests. Of these 1 had sprue, 3 had regional enteritis, and 2 had Whipple's disease (see Table I and II).

A group of 44 patients clinically classi-

Table II

RESULTS OF THE BLOOD STUDIES

	Number	Abnormal	Per Cent Abnormal	3 to 6 hr. Average Blood Level Per Cent
Normal	33			12 to 13
Functional diarrhea	44	0	0	12 to 13
Chronic pancreatitis	12	10	83.3	3 to 4
Carcinoma of pancreas	19	18	94.7	3 to 4
Hofmeister resection	48	23	48.0	8 to 9
Vagotomy and gastroenterostomy	22	16	72.8	6 to 7
Billroth 1 resection	12	5	41.0	9
Whipple's disease	2	2	100	5 to 6
Sprue	1	1	100	1 to 2
Regional enteritis	3	3	100	3 to 4

fied as having functional diarrhea was tested and in each instance normal values were obtained.

DISCUSSION

The use of an I^{131} glycerol trioleate test meal provides a practical, effective and simple means of evaluating digestion and absorption of fat. The uniform reproducible results obtained in control studies make it apparent that variations from these normal values should be of clinical significance. Although the fat test in itself may not lead to a specific diagnosis, an abnormal test, when coupled with other clinical and laboratory information, may point the way to the correct conclusion.

In the laboratory the test can be conducted in several ways; either the blood or the fecal radioactivity may be determined, or the combination of the two obtained. The close correlation between the blood and fecal values has been impressive. In every instance when the blood level has been low, the fecal radioactivity has been significantly elevated. Our experience has been that the fecal determinations are the most valid since they represent the recovery of that part of the test meal which has not been absorbed. A combination of the blood and fecal determinations, however, represents the best plan of study for it provides a cross-check on the data obtained. In several instances only the blood or the fecal specimens were analyzed which accounts for the apparent discrepancies in correlation of these values.

From the practical standpoint, the fecal fat determinations are simpler than the blood determinations. They can be carried out in any laboratory equipped to do rou tine thyroid uptake studies. The fecal samples arrive in individual, unopened containers and can be evaluated for radioactivity by external counting.[4] Blood samples, on the other hand, must be counted in a scintillation well counter.

It is of importance to know to what the radioactive material is tagged in both the blood and the feces. Dialysis studies and electrophoretic determinations have shown that the I^{131} which first appears in the blood is bound to the beta lipoprotein and neutral fat fraction. The radioactive material in the feces is nondialyzable, and appears in the fat fraction when extracted by chemical techniques. These facts afford the proof that the radioiodine is bound to fat and is not initially present in the blood as free iodine or in the feces at any period as free iodine. This information indicates that the fecal test, particularly, represents a type of fat balance study.

The control studies made it apparent that the rate of gastric emptying influences the initial blood levels. In order to keep a check of the gastric phase of emptying, we have routinely followed the progress of the test meal in each patient, either by fluoroscopy or by appropriate roentgenograms. Without such control, low blood levels of radioactivity might be misconstrued as evidence of faulty absorption or digestion.

The fat test, when applied to patients with pancreatic disease, has provided consistent results. The blood levels have been considerably lower than normal and fecal fat levels have been impressively higher than normal.[5] Pancreatic lesions in order to give an abnormal test must either block the duct and thus prevent discharge of enzymes into the intestinal tract or so alter function of the acini that enzymes are not produced. The patient with carcinoma who gave a normal test was found at operation to have a lesion in the tail of the pancreas and the enzymes produced were therefore able to reach their destination. The 2 patients with nonmalignant pancreatic disease whose tests were normal demonstrated the obvious fact that there is considerable reserve function in the pancreas and that in these 2 instances enzymes were both produced and discharged into the intestinal lumen. It has been our experience thus far that a positive test, characterized by high fecal fat and low blood levels coupled with the roentgen demonstration of a normal intestinal tract is most indicative of pancreatic disease. Recently, in several

cases studied just such a situation led to a preoperative impression of pancreatic disease in spite of certain evidences indicating otherwise, and at operation carcinoma of the pancreas was demonstrated.

Another practical aspect of the fat test lies in its application to the evaluation of fat absorption and digestion following various surgical procedures for peptic ulcer. A high percentage of the cases studied demonstrated by the test the abnormality in fat uptake[3] similar to that found by others using chemical methods.[2] Radioisotope technique can be used to quickly and simply establish the status of fat uptake, and the fact that the test can be repeated on numerous occasions allows one to evaluate the effects of various treatments and dietary supplements. Thus, a relatively simple and accurate method is made available to gauge the efficacy of treatment.

Since clinical improvement is often delayed during treatment, the fat test might prove decisive as to whether to continue or discontinue a given mode of therapy. Demonstration of a rise in blood levels after treatment indicates strongly that the treatment may prove of value, even in the absence of initial clinical response.

This aspect of the test also applies to those patients with primary small bowel abnormalities. Again in such cases we may get information concerning the value of treatment prior to obvious clinical improvement. In one such case studied, a previously abnormal test reverted toward normal when therapy was instituted. Clinical evidence of improvement became manifest several weeks later. The test likewise may help in another way by indicating impending clinical signs and symptoms. On several occasions we have found markedly elevated fecal fat values in asymptomatic patients who later developed significant symptoms.

A significant feature of the study concerns the group of patients with so-called functional diarrhea. In each of these individuals no abnormal anatomic diagnosis could be established in spite of many studies. The fact that in all instances following the administration of the tagged fat meal normal values were obtained provided proof that diarrhea in itself does not lead to an abnormal test. Moreover, the normal values obtained may aid in properly evaluating the clinical status of such patients.

We have emphasized in this presentation the practical aspects of the radioactive fat test meal. There are other equally important features of the technique which we wish to stress. It provides a new approach to the study of gastrointestinal physiology, and such studies have been and are still in progress. They will be reported upon at a later date. Also, other tagged fats may prove of value and are now under investigation. In the meantime, however, the test offers a practical advance in the evaluation of the absorption of fat in the gastrointestinal tract and has proved helpful in a variety of clinical situations.

CONCLUSIONS

1. I^{131} glycerol trioleate has been utilized in the study of digestion and absorption of fat.

2. Normal subjects give uniform reproducible levels of radioactivity in both blood and feces under the conditions of the test.

3. Application of the test to groups of abnormal patients has shown that the test can be of clinical value in pancreatic disease, postoperative surgical patients, and in diseases of the small intestine.

4. The fat test meal may serve as a guage or barometer in determining the effects of certain forms of treatment supplements upon fat digestion and absorption.

5. Fecal recovery of radioactivity as conducted constitutes a new type of fat balance study.

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HEPATOGRAPHY

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CONSIDERABLE need exists for the development of safe practical methods for roentgen examination of the internal structure of the liver. Such a technique could prevent unnecessary operations upon patients with metastatic carcinoma of the liver. Solitary metastasis could conceivably be excised if the exact size and location within the liver bed could be determined. Needle and surgical biopsy of the liver could be greatly improved in accuracy if the exact location of lesions could be located. Non-neoplastic lesions, such as cysts and abscesses, could be diagnosed and localized for biopsy, drainage or removal. A new procedure would be added for the detection of metastasis in postoperative cancer patients.

Attempts to fulfill the above need have taken several forms:

(1) Injection of radioactive materials in the hope of obtaining hepatic tissue uptake, without uptake by metastasis, or metastatic tissue uptake without parenchymal uptake, and the graphing of results by means of scintillating counters.
(2) Arterial injection of radiopaque media by catheter through the abdominal aorta.
(3) Venous injection of portal vein by way of percutaneous injection of the spleen.
(4) Attempts to obtain better contrast by pneumoperitoneum.
(5) The developing of contrast between liver and metastasis by the production of radiopacity within the liver parenchyma.

All of the above methods to date have had the disadvantages of either poor or no delineation of the lesions deep in the liver parenchyma, visualization of primarily venous or arterial tree or toxicity of the administered materials. Many materials have been tried in an attempt to obtain satisfactory opacification of the hepatic parenchyma. Thorium dioxide suspension (thorotrast) gives excellent permanent opacification of liver and spleen with little immediate adverse reaction. However, reports of suspected chronic toxicity in the form of hepatocellular degeneration, cirrhosis, and late carcinoma have appeared. While not numerous these reports will probably prevent the material from becoming popular except in certain selected cases. Jodosol (triiodostearate) and other iodinated compounds have been used.[2,9,13,20,25,26,38] Brominated sesame oil has been employed.[19] None of these materials give truly good opacification and all have immediate toxicity.

In general the standard techniques for taking plain roentgenograms of the abdomen have been used with an attempt to visualize nonopacified metastasis against the entire mass of opacified liver cells. Resultant films have usually shown poor delineation of small lesions and poor localization of lesions as to site and depth.

We wish to present a technique which, while not getting away from potential toxicity of the opacifying media, does permit excellent visualization and localization of small metastases. This technique, in addition, requires no special laboratory or equipment as it is accomplished with roentgenographic equipment standard in many hospitals.

TECHNIQUE

Laminagraphy is performed after opacification with thorotrast. Twenty-five cc. of colloidal thorium dioxide in 25 per cent

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solution is injected intravenously on each of three successive days, and twenty-four hours after each injection the liver and spleen are roentgenographed to determine the degree of contrast. When sufficient contrast has been developed, serial laminagraphy at 1 cm. intervals is carried out in the posteroanterior projection. It has been found that 200 ma. at 2 seconds with a kv. range between 62 and 76, depending on patient thickness, gives the most satisfactory outlining. In very obese patients or in the presence of a markedly enlarged liver and/or ascites, the ma./seconds must be reduced to decrease haziness due to scatter. In these cases it is necessary to use a higher kv. (between 80 and 88 with 100 ma. at 2 seconds).

RESULTS

Defects due to metastasis can be outlined with accuracy at various depths. Even after 25 cc. of thorotrast, these defects could be faintly visualized but after 75 cc. the outlines of these defects, indicating absence of liver tissue, were easily seen and sharply delineated. Accurate localization as to depth and site of metastasis has been accomplished easily in all patients. Metastases of the size of 1 cm. or less in the depth of the liver parenchyma have been consistently detected. These points have been confirmed by postmortem studies with laminagraphy of the liver, mechanical slicing of the liver, photographs, as well as autoradiographs. There has been no instance of "missed metastasis."

Fourteen patients were studied in the initial series. All had biopsy proved carcinoma in an inoperable stage, and suspected or proved hepatic metastasis. There were two minor reactions to the thorium injections; both were chills and fever without shock or other disturbances. These reactions did not necessitate stopping the procedure. Visualization of the liver and metastasis was accomplished in all patients.

REPORT OF CASES

Case 1. P.J.A., a sixty-seven year old white male, was admitted with an eight week history of dull epigastric pain. A huge epigastric mass and palpable left axillary lymph nodes were present. Biopsy of the lymph nodes revealed anaplastic carcinoma. A primary source was not found and the prothrombin time was too low to permit biopsy of the liver. Laminagraphy after thorotrast injection showed a markedly enlarged liver, involving both lobes. The lingular lobe projected beyond the midline and downward. Numerous areas of radiolucency were demonstrated in the liver, especially the lingular lobe, indicating destruction of liver tissue due to multiple metastases. The spleen also was visualized and was enlarged. Patient continued relatively asymptomatic on supportive measures.

Case II. D.I.St.J., a sixty-five year old white male, gave a history of right pleural effusion. A positive Papanicolaou smear of sputum and positive cell block of pleural fluid for neoplasia were obtained. He received radioactive gold intrapleurally with a fair response. Hepatography showed both lobes of the liver markedly enlarged with numerous areas of radiolucency indicative of metastases concentrated mainly in the upper portion. The patient remains in the hospital on symptomatic care.

Case III. E.A., Jr., a forty-three year old colored male, had previous surgery in May of 1954 for carcinoma of the stomach. Ascites was

Fig. 1. Case III. Hepatography (laminagraphy following intravenous injection of thorotrast) showing well-defined areas of radiolucency indicating metastatic replacement of liver tissue.

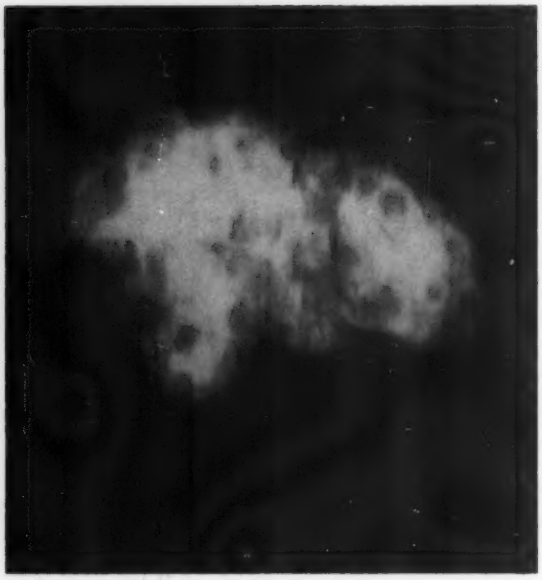

Fig. 2. Case III. Laminagraphy of postmortem liver specimen with cut at about the same level showing similarity of lesions.

noted on re-admission, but the liver was not definitely palpable. Hepatography showed enlargement of both lobes of the liver with numerous areas of radiolucency. The greater portion of the liver substance appeared to be replaced by metastatic carcinoma with only a shell of liver tissue remaining. The patient continued a progressive downhill course until death. Autopsy revealed multiple carcinomatous nodules in the liver, metastatic from the stomach. These hepatic nodules had a distribution identical to those seen on laminagraphy. Laminagrams of the liver specimen which was examined separately showed well outlined metastatic nodules which matched the appearance of the liver *in situ*. The splenic shadow visualized well and appeared small.

Case IV. C.E.H., a sixty-two year old white male, had a one year history of abdominal pain and weight loss. Chronic cough had been present for years. He gave a history of chronic alcoholism. Abdominal distention was noted but no organs were palpable. Chest roentgenogram revealed a mediastinal mass. Bronchoscopy showed a bronchial mass which could not be reached for biopsy. Progressive liver enlargement occurred. Because of the poor risk, a liver biopsy was deferred and hepatographic laminagraphy with thorotrast was done instead. The patient expired before examination was completed. Laminagraphy after only one injection showed faint visualization of both liver and spleen with numerous large areas of radiolucency involving both lobes of the liver, which were interpreted as large metastatic nodes. Autopsy revealed a carcinoma of the lung with metastasis to the liver. Cirrhosis of the liver was present. The liver metastases were huge and involved the entire liver, including the right lingular lobe, as indicated roentgenographically.

Case V. D.D.H., a sixty-eight year old white male, had squamous cell carcinoma of the sigmoid colon diagnosed microscopically after resection of the colon in 1953. He had a sudden onset of abdominal pain and nausea. Six months later a nodular mass was felt in the liver.

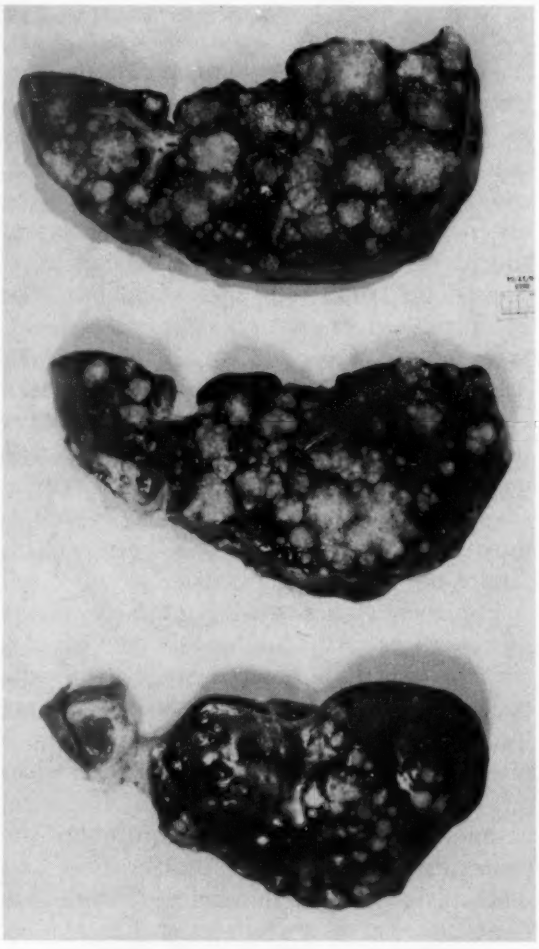

Fig. 3. Case III. Photograph of sliced liver showing the extensive metastasis and the similarity of appearance to the premortem hepatography.

Fig. 4. Case V. Hepatography showing enlargement of both lobes of liver and huge destructive lesions in the right lobe, best seen at the 12 cm. level.

Laminagraphic studies of the liver with thorotrast showed enlargement of both lobes of the liver. The lower portion of the right lobe outlined as a huge area of radiolucency with a thin rim of opacified liver tissue, indicating the presence of a large metastasis which almost entirely destroyed the liver. The lingular lobe showed numerous areas of radiolucency due to metastasis. The splenic shadow was slightly enlarged. Follow-up examination two months later showed considerable increase in the destruction of the liver with almost complete transformation of the liver into a large metastasis as evidenced by large areas of radiolucency. The patient expired four months after admission. Autopsy confirmed the roentgen findings and distribution of the squamous cell carcinoma. Laminagraphic examination of the liver after necropsy gave an appearance identical to that found pre mortem.

Case vi. E.A.R., a fifty-nine year old white male, had a six week history of upper abdominal fullness of a progressive nature and loss of 20 pounds weight in six weeks. An epigastric mass had been present for four weeks. There was a huge hard irregular epigastric mass and a tender fluctuant mass in the left flank. Rectal examination disclosed a small hard nodule submucosally. Biopsy of the flank lesion revealed an abscess. Needle biopsy of the liver showed carcinomatous tissue without any visible liver tissue. The primary source could not be determined. The patient became jaundiced and developed marked edema and ascites. Hepatography confirmed the marked enlargement of the liver, as seen clinically. The entire liver tissue appeared to be replaced with numerous areas of metastasis, some of which measured more than 12 cm. in diameter. Both lobes showed almost complete destruction by

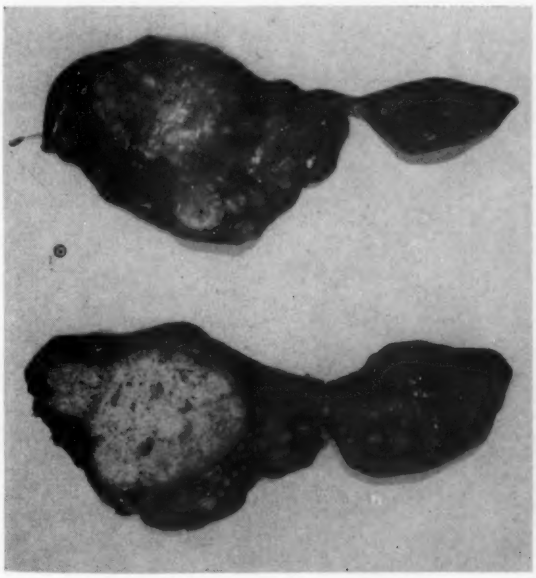

Fig. 5. Case v. Photograph of sliced liver specimen confirming the premortem roentgenographic appearance.

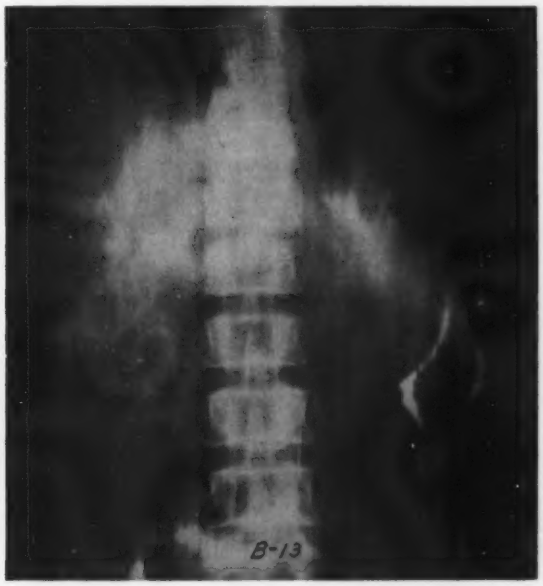

Fig. 6. Case vi. Hepatography showing marked enlargement of the liver with almost complete replacement of liver tissue by metastatic carcinoma.

Fig. 7. Case vii. Hepatography showing the enlargement of liver with numerous metastatic nodules replacing liver tissue. The great number of nodules prevents distinct outlining of the individual lesions.

metastasis. This patient also had a follow-up examination two weeks later and this showed progression of the replacement of liver tissue by metastasis. The patient expired after massive bleeding per rectum. Autopsy revealed adenocarcinoma of the liver with pulmonary metastasis, and confirmed the roentgen appearance of almost complete destruction of the liver.

Case vii. O.E.W., a sixty-three year old white male, had lower abdominal pain for three months, weight loss of 15 pounds and clay colored stools for an unknown period. Icterus was present as was gross liver enlargement. A nodular mass was present in the right upper quadrant near the midline. Needle biopsy of the liver revealed a columnar cell carcinoma. Hepatography showed both lobes markedly enlarged. Numerous small to moderately enlarged nodular areas of radiolucency involved the parenchyma of both lobes indicating almost complete destruction of liver tissue. They were so numerous that distinct outlines were difficult to obtain. The patient had a downhill course and expired. Autopsy revealed a columnar cell carcinoma of the gallbladder with metastasis to liver, lymph nodes and lungs. Metastases were extremely numerous, replacing nearly all normal hepatic parenchyma, confirming the premortem roentgenographic findings. Laminagraphic examination of the liver specimen after necropsy outlined the nodular areas of radiolucency with greater detail and showed almost complete replacement of the liver parenchyma by the metastases. Autoradiographs of slices were also done and further confirmed the roentgen findings.

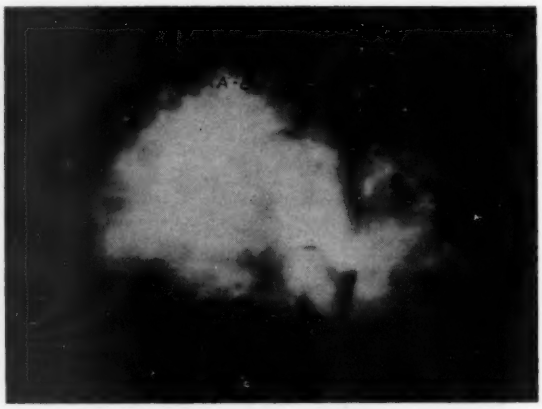

Fig. 8. Case vii. Postmortem laminagraphy of liver at about the same level showing the numerous metastatic nodules.

Case viii. J.B.S., a sixty year old white male, was admitted with a three month history of weight loss and anorexia. The liver was markedly enlarged, firm and slightly tender with

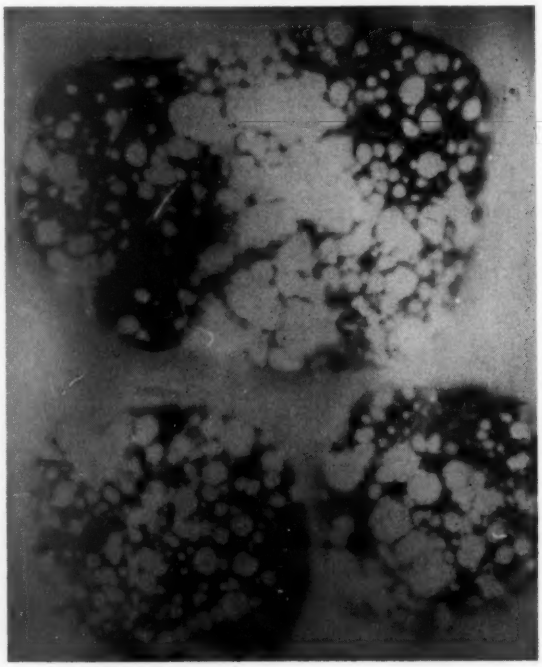

Fig. 9. Case vii. Autoradiograph of the sliced liver showing almost complete replacement of hepatic tissue by the metastatic nodules.

irregular borders. Chest roentgenogram revealed a hilar mass. A needle biopsy of the liver disclosed carcinoma. Hepatography showed such extensive metastatic involvement of the liver that there was insufficient normal parenchymal tissue to absorb the radiopaque thorotrast. The liver outlined as numerous indistinct areas of radiolucency. The patient became progressively comatose and expired. Autopsy revealed carcinoma of the left lung with metastasis to regional and retroperitoneal lymph nodes and liver. Practically no liver parenchyma remained, confirming the laminagraphic findings.

Case ix. R.C., a forty year old colored male had a long history of alcoholism, developed epigastric pain and swelling four weeks prior to admission. Examination revealed marked hepatomegaly with a tumor mass palpable in the epigastrium. A systolic murmur was heard over this mass. Surgical biopsy disclosed a vascular tumor diagnosed histologically as a primary liver cell carcinoma. Hepatography confirmed the marked enlargement of both lobes of the liver with well delineated areas of metastasis in both lobes. The lingular lobe appeared to be almost entirely replaced by a large area of radiolucency which represented a large metastatic node. The patient expired after a slow downward course. Autopsy revealed liver cell carcinoma at precisely the sites demonstrated by hepatography.

Fig. 10 Case ix. Hepatography showing well delineated large areas of malignant tissue replacing the hepatic tissue with marked enlargement of the liver.

Case x. W.D.U., a sixty-four year old white male, had excision six years previously of a basal cell carcinoma of the nose. Local metastasis occurred to the cervical lymph nodes. He was admitted with an unassociated cylindroma of the penis diagnosed on biopsy. Hepatomegaly was noted. Hepatography confirmed the clinical impression of a large liver and showed numerous areas of radiolucency with well-defined metastatic nodules. Postmortem examination confirmed the presence of basal cell carcinoma, metastatic to the neck, and a malignant cylindroma of the penis with metastasis to the liver. The configuration of the metastasis was identical to that demonstrated on the laminagrams.

Case xi. J.F.H., a fifty-nine year old white male, had a previously biopsy proved diagnosis of squamous cell carcinoma of the lung, with metastasis to the cervical lymph nodes. The liver was not enlarged. The patient expired apparently of progressive bronchial obstruction. Autopsy was not permitted. Premortem hepatography showed the liver within normal limits in size but with moderate nodularity of its borders, with occasional areas of radiolucency, suggestive of metastatic destruction of liver parenchyma.

Case xii. C.N., a sixty year old white male, had biopsy proved carcinoma of the lung for which he received local irradiation in 1951. He had done well since, but developed progressive hepatomegaly. He had a history of alcoholism. The differential diagnosis of the liver enlargement was between cirrhosis and metastatic carcinoma of the liver. Hepatography outlined the liver and showed it to be slightly enlarged with lobulation. Faint areas of radiolucency indicative of small metastases were demonstrated on the laminagrams. The patient continues well with some weight loss.

Case xiii. F.P., a forty-two year old white male, had a six weeks' history of pneumonia followed by shortness of breath, hoarseness and weight loss. A Horner's syndrome was present. Biopsy of a right supraclavicular lymph node confirmed the diagnosis of carcinoma of the lung. The liver became markedly enlarged but was not tender. Chest roentgenogram revealed an irregular mass in the left upper lobe. Hepa-

tography confirmed the marked enlargement of both lobes of the liver with numerous well defined and well demarcated areas of decreased density indicative of numerous metastases. The lingular lobe was markedly enlarged and extended well into the left abdomen. It was almost completely replaced by metastatic nodules. The lesion in the left upper lobe was irradiated and the patient discharged asymptomatic.

Case xiv. A.B., a forty-eight year old colored male, gave a history of left upper abdominal pain for one year and suffered a 30 pound weight loss in the same period. The liver was enlarged, firm and nodular. A hard nodule was present in the abdominal wall and a Blumer's shelf palpable in the rectum. A roentgenogram of the stomach revealed carcinoma. Needle biopsy of the liver showed metastatic columnar cell carcinoma. Hepatography confirmed the clinical impression of enlarged lobes of the liver. Several well-defined areas of radiolucency were present in both lobes of the liver indicative of metastatic destruction of the liver parenchyma. The patient did well on symptomatic therapy and even gained some weight. No surgery was performed.

Discussion

The technique as described gives excellent results and accurately localizes metastasis within the liver parenchyma. It would seem that two of the major obstacles to an adequate clinical roentgenologic technique have been overcome. These are (1) adequate visualization of small metastases, and (2) accurate localization of metastases within the liver parenchyma. It is to be pointed out that while only posteroanterior laminagraphy has been done, lateral laminagraphy may also be performed for further localization.

Two or more major obstacles remain. These are (1) lack of an absolutely safe contrast medium, and (2) our probable inability to distinguish between benign and malignant space occupying lesions in the hepatic parenchyma. Additional experience may aid in this differentiation.

Radioisotope techniques have been proposed in the solution of this problem. These materials (iodinated albumin, radiogold and radiochromate) add problems of their own. In general, radioisotopes demonstrate peripheral metastases and fail to reveal lesions deep in the liver. Radioisotopes with short half-lives are probably safe enough. Localization of tumors is generally poor unless special three dimensional techniques are used, necessitating highly specialized equipment and highly trained personnel with the use of radioisotopes.

Visualization of the vascular structure of the liver and spleen by aortography or splenography provides at times satisfactory delineation of the vascular tree but fails to demonstrate small parenchymal lesions. The procedures carry some risk and again require highly trained personnel.

In its present form our technique has been limited to the patient with proved inoperable primary carcinoma and proved or highly suspected metastatic disease of the liver. We are slowly expanding the scope of our study and would like to suggest the following conservative indications and contraindications.

1. General Contraindications
   - A. Patients under sixty-five years of age.
   - B. Patients who do not have proved primary carcinoma.
2. Indications
   - A. Patients with biopsy proved carcinoma and hepatomegaly or other indications of hepatic metastasis (elevated alkaline phosphatase, for example).
   - B. Prior to extensive resectional surgery in aged patients with proved primary malignant neoplasms of the abdomen and without clinically demonstrable secondary liver metastases.
   - C. Older patients with suspected hepatic metastasis whose condition is so poor that extensive study and exploratory laparotomy are not feasible.
   - D. All patients with biopsy proved primary carcinoma of the liver.

As with all procedures in medicine the

potential dangers must be weighed against potential benefits. The indications above were chosen with due regard for the toxicity of thorium but also with consideration for the adverse effects of laparotomy and other vigorous diagnostic procedures in inoperable carcinoma. Roentgenography in primary carcinoma of the liver offers the hope of discovering localized tumor which can be resected.

Were a safe contrast medium available for hepatic visualization, the scope of this technique could be greatly expanded to include routine hepatography of all cancer patients. Benign lesions might then be localized for drainage, biopsy, and therapeutic evaluation.

Our present studies involve screening procedures in animals in an attempt to find an adequate contrast medium. To date we have failed to liberate thorium from the liver with chelates and BAL. The use of brominated sesame oil has been disappointing. Our current attempts are to use insoluble organic iodides ground to 1–10 micra and injected intravenously.

SUMMARY

Laminagraphy of the liver with the use of preliminary intravenous injection of thorium dioxide provides excellent visualization and localization of hepatic neoplasms, whether primary or secondary. The limiting factor in the use of the technique is fear of delayed reactions due to thorium dioxide.

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RENAL ESCHERICHIA COLI INFECTION ASSOCIATED WITH DIABETES MELLITUS

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DIABETES mellitus produces an inherent susceptibility to infection, especially of the skin and urinary tract. Infections of the urinary tract are caused by coliform bacilli in about 80 per cent of the cases, and most of the remainder are caused by Staphylococcus aureus. Escherichia coli infection, associated with diabetes, is known to occasionally produce spontaneous gas by fermentation of sugar. The first case presented here is one where spontaneous gas was produced in the renal and perirenal tissues. The second case presented is that of necrotizing papillitis, a pathologic entity that occurs in about 25 per cent of diabetics who die with pyelonephritis. It is also known that about 20 to 27 per cent of diabetics have pyelonephritis at the time of death, as has been shown by several series of autopsies.

Spontaneous renal emphysema and necrotizing papillitis are highly fatal entities if they occur in both kidneys, but recovery may follow if the disease process is confined to one kidney, as occurred in both of our cases. Both conditions are seen most frequently in diabetics and nearly always are caused by coliform bacilli but occasionally various other bacteria are found. In the cases presented here, diabetes was present and Escherichia coli was cultured from the urinary tracts, but in the case of renal emphysema there was obstruction or at least stasis of the urine. Both lesions are rather rare, although it is probable that the necrotizing papillitis does occur more often than realized. Their roentgen signs are easily recognized, and it is possible that by earlier roentgen diagnosis of renal papillitis, some lives may be saved. The similar etiologic factors and the paucity of reports in the literature as well as other aspects of both cases are reasons for reporting them.

Spontaneous production of gas by bacterial infection has been well recognized and usually thought of as gas gangrene produced by Welch's bacillus, a clostridial infection. Consideration of the nonclostridial gas producing infections by aerobic and anaerobic bacteria is often neglected. The aerobic bacteria which may produce gas are Escherichia coli and pyogenic cocci, and the anaerobic bacteria are the cocci and the Bacteroides group. The Escherichia coli is by far the most important producer of gas by fermentation of sugar and may produce gas in the tissues of poorly or uncontrolled diabetics or in nondiabetic patients, usually if they have received a large amount of glucose in injected fluids. These nonclostridial gas producing infections may occur in the amputated stump of diabetics, where they must be differentiated from Welchii infection. They may also be encountered in the abdomen, as for example in the gallbladder and pancreas, and in the urinary tract.

Gas producing infection of the urinary tract was first described in the urinary bladder in 1825 by Boismont. Until 1860, most of the cases reported were caused by instrumentation or fistula, but at that time, Raciborski reported a case of spontaneous development of gas in the bladder, and named this condition pneumaturia. In 1883, Guiard first associated pneumaturia with diabetes and ten years later, in 1893, Chiari reported a case of pneumaturia caused by Escherichia coli in a diabetic. Other cases have been reported of other bacteria causing pneumaturia, with or without the association of diabetes, but most of the cases have been in diabetics and caused by Escherichia coli. Another form of spontaneous production of gas in the urinary tract occurs in the bladder and is called cystitis emphysematosa, when the bladder wall contains cysts. The first re-

port of roentgen evidence of spontaneous pneumaturia was by Mulsow and Gillies in 1934.[11]

In 1938, Shields Warren[19] mentioned in his book, "Pathology of Diabetes," that he had seen a roentgenogram showing air in the renal and perirenal tissues outlining the kidneys, and this is the first reference of roentgen evidence of this condition occurring in the kidneys.

One year later Olsson[12] reported a case of diabetes with Escherichia coli infection of the urinary tract producing necrotizing papillitis, pyleonephritis, and spontaneous gas in the urinary tract, which resulted in a gas pyelogram.

Gillies and Flocks[6] in 1941, gave a comprehensive discussion of this condition from a roentgenologic viewpoint. Their case of spontaneous emphysema of the renal and perirenal tissues occurred in a poorly controlled diabetic with recurrent urinary tract infections, associated with obstruction of the urethra by a cyst or abscess of Gartner's duct, producing obstruction of the bladder and subsequent ascending infection of the renal and perirenal tissues. The infecting organism was coliform bacillus, and they postulated that this condition was caused by three factors: diabetes, colon bacillus and lower urinary tract obstruction. It is believed that lower urinary tract obstruction produces ascending infection of the upper urinary tract more often than ureteral obstruction. In the case of Gillies and Flocks, both kidneys were involved and death resulted.

No case has been previously reported associated with pregnancy.

REPORT OF CASES

Case 1. A nineteen year old woman entered Memorial Hospital of South Bend, Indiana, in her thirty-second week of pregnancy. She was known to have had diabetes since three years of age. During the pregnancy, she had frequent medical care for recurrent urinary tract infection and for difficulty in controlling her diabetes. Her blood sugar and urinary sugar were high most of the time. Shortly after admission to the hospital, roentgenograms of her abdomen (Fig. 1) in various positions for the pregnancy revealed nothing unusual and no gas or other changes were present around either kidney area.

A cesarean section was done with delivery of a live baby, but postoperatively, she had a spiking fever and abdominal distention and nausea. Her urine had contained pus during most of the pregnancy and also during the postoperative period. Escherichia coli was cultured from the urine several times.

Eleven days postoperatively a examination of the urinary tract (Fig. 2) revealed a large amount of gas ballooning out the subcapsular renal space, with numerous radial streaks throughout the left kidney substance. The gas also passed out beyond the capsule in the perirenal area with multiple various sized gas bubbles extending mostly in a caudal direction. An intravenous pyelogram revealed no function of the left kidney. The right kidney was normal in function and outline. The gas distribution throughout the kidney seemed to follow the interstitial tissue similar to that of the glycogen deposits in Henle's loops, convoluted tubules and capsular space as described in diabetes by

Fig. 1.

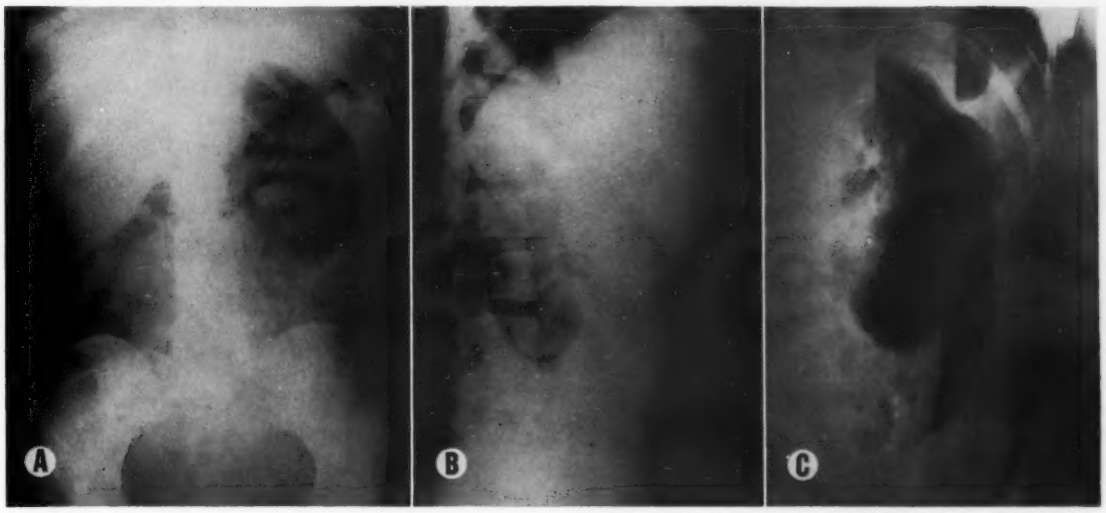

Fig. 2 (*A*, *B* and *C*).

Shields Warren. Since Escherichia coli does not ferment starch, there is probably also glucose in high concentration present in these cells in the process of resorption from the tubules. Harada has found that there is a fair degree of correlation between urine sugar and the amount of glycogen infiltration of the kidney.

An operation to establish drainage was done and multiple abscess pockets were found surrounding the left kidney. A culture of this pus revealed pure Escherichia coli. Several months later, she was complaining of diarrhea, with some passage of blood from the bowel, and injection of radiopaque material revealed a sinus tract connecting the left renal pelvis with the colon.

After about one year, the patient recovered sufficiently so that the fistulous tract was closed and, at this time, the infection had been so destructive, that the kidney could not be identified at the time of surgery.

In this case, in contrast to others reported, there was only one kidney involved so that recovery was possible. This, then, represents a case of spontaneous renal and perirenal emphysema demonstrated roentgenolgoically with apparent complete destruction of the kidney involved.

Case II. This was a case of Escherichia coli infection of the kidney occurring in a patient known to have had diabetes for eight months. This fifty-seven year old woman had frequent attacks of spiking fever and malaise, and her diabetes was difficult to control. She had an elevated sedimentation rate with a higher insulin requirement than thought necessary, but the urine microscopically on several occasions did not indicate urinary tract infection. It is known that urinary tract infections are often missed or neglected in spite of the fact that they are present frequently in diabetics.

It was not until after five months of repeated attacks of fever and malaise that pyuria was discovered and culture of the urine revealed Escherichia coli. Sensitivity tests were done and the indicated antibiotics were given without benefit. An intravenous urogram (Fig. 3), taken at the time of discovery of the diabetes, showed normally functioning kidneys and no deformity of the calyces or any part of the urinary tract.

Eight months later, another intravenous urogram was made (Fig. 4) and at this time, there was a poorly functioning left kidney with a pooling of the contrast medium in multiple pockets, giving a sort of moth-eaten appearance. A retrograde pyelogram of the left kidney (Fig. 5) showed more clearly the pockets of contrast medium around the calyces, simulating extravasation of the material. According to the literature, the early roentgen changes in this condition may be only those of beginning pyelonephritis. As demarcation and separation of the papillae develop, minor deformities occur in the calyces, and as the disease advances, ring shadows form and filling defects occur with the sloughing off of the papillae. When the necrotic areas have sloughed off and pass into the renal pelvis, filling defects in the contrast-filled renal pelvis may be seen on urographic examination.

Fig. 3.

It has been reported that fragments of the loose necrotic tissue have been recovered in the urine. If the fragments of tissue can be found and recognized in the urine, the diagnosis becomes apparent.

In this case, the kidney was removed and showed multiple pockets in the pyarmid areas of the kidney with little inflammatory reaction (Fig. 6).

Grossly and microscopically, the pathologist reported a chronic pyelonephritis and an acute necrotizing papillitis, with necrosis of the papillae and some associated inflammatory changes of the medulla. The necrosis was of the coagulative type. There was no evidence of diabetic intracapillary glomerulosclerosis of nephritis. Culture of the pus from the kidney showed Escherichia coli. This pathologic entity may be present in various stages of separation of the papillae within the same kidney in various pyramids of the kidney.

Von Friedreich, in 1877, first described renal papillitis. Then Gunther, in 1937, renewed interest in this lesion by describing the pathologic entity of renal papillitis or necrotizing papillitis and its association with diabetes.

There are several theories as to the etiology of necrotizing papillitis, but it is most commonly thought to be the result of interference with the vascular supply by a fulminating infection such as may be observed with Escherichia coli. It is more common in diabetes, usually occurring in women over forty years of age, who do not have urinary tract obstruction. It also occurs in men, generally when they are over sixty years of age and have prostatic obstruction of the urinary tract but are not necessarily diabetics. This not so well known entity is highly destructive to the kidney and fatal to the patient if both kidneys are involved. In both diabetics and nondiabetics, about one-third of cases have unilateral involvement. The course of the disease may be acute or chronically progressive.

Case 11 made a prompt recovery postoperatively. The sedimentation rate returned to normal and the insulin requirements were reduced to one-half of the dose before surgical removal. In this case, the roentgenogram called attention to the destructive inflammatory change in the kidney and was the deciding factor for surgical removal, with resulting recovery of the patient, who has remained well for the past ten months.

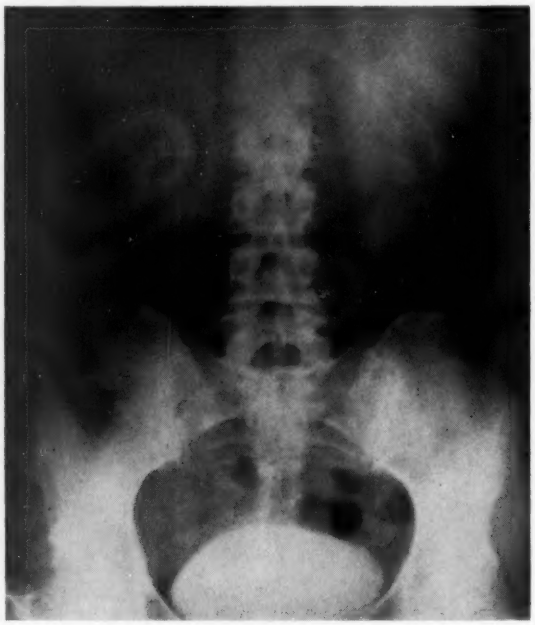

Fig. 4.

DISCUSSION

Cases I and II are of interest because of similar etiologic factors but producing two different lesions in the kidney, one spontaneous emphysema and the other necrotizing papillitis. Both patients were diabetics and both had repeated urinary tract infections caused by Escherichia coli. In one there had been obstruction or stasis of urine due to pregnancy, but no obstruction was present in the case of the necrotizing papillitis. It is also known from previous reports in the literature that neither diabetes nor Escherichia coli is necessary for either lesion to occur, although both play a great role in their pathogenesis. Gillies and Flocks,[6] thought that it was necessary to have diabetes, Escherichia coli infection and obstruction of the lower urinary tract for the production of spontaneous renal and perirenal emphysema, but in the case presented here, the stasis or obstruction of pregnancy is usually considered to involve the ureters and upper urinary tract above the bladder. Also, in Case I the demonstration of spontaneous emphysema occurred in the postpartum period.

FIG. 5.

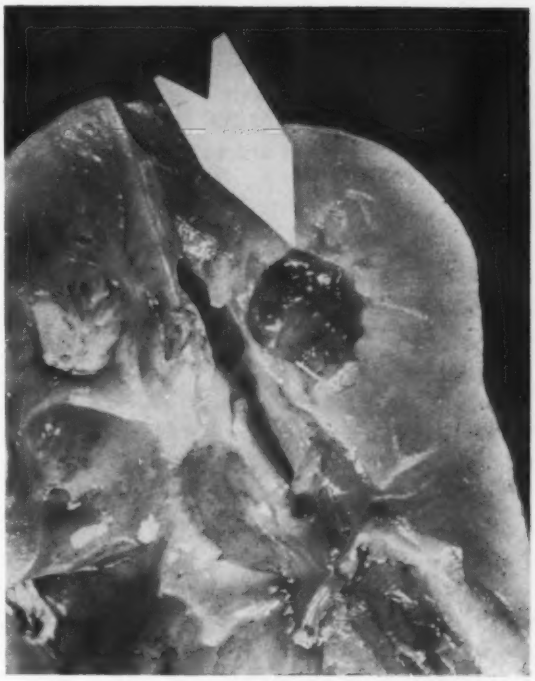

FIG. 6.

It is known that insulin requirements rise in about 75 per cent of diabetics after the third month of pregnancy with the diabetes being more difficult to control. It is likely that more sugar is retained in the kidney. When one considers that this fact alone creates an ideal medium for the production of gas by the coliform bacilli in a diabetic and for the production of necrotizing papillitis, it is surprising that many more cases do not occur. The very destructive effect of both lesions to the kidney is rather striking. The inciting cause precipitating the lesions is not known and although many cases of diabetes have similar backgrounds, in only a few does either lesion occur. Pyelonephritis seems to be a forerunner of necrotizing papillitis and its recognition as early as possible may reduce the incidence of this lesion. One wonders about the possibility of necrotizing papillitis being necessary for the development of spontaneous renal emphysema. Both lesions have separate characteristic roentgen appearances. Urinary tract infection in diabetics is fre-

quent but usually asymptomatic or obscure. Therefore, by more frequent intravenous urography in diabetics, it may be possible to reduce the mortality of the highly fatal renal papillitis by earlier recognition and proper treatment.

CONCLUSIONS

1. Spontaneous renal and perirenal emphysema and necrotizing papillitis occurred in 2 cases of diabetes with similar etiologic factors.

2. Escherichia coli infection in poorly controlled diabetes was present in both cases.

3. Spontaneous renal and perirenal emphysema occurred in the postpartum period of pregnancy.

4. Renal papillitis occurred in a woman with diabetes, where evidence of the presence of infection was present, but not localized to the urinary tract until five months after onset.

5. Both lesions produced marked destruction of the kidney involved, but both cases recovered since only one kidney was involved in each instance.

6. Closer observation and proper treatment of the urinary tract in diabetics may result in prevention or possible cure in some cases of renal papillitis.

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USE OF SENNA FOR PREPARATION OF THE COLON FOR ROENTGEN EXAMINATION*

By S. A. KAUFMAN, M.D.
BOSTON, MASSACHUSETTS

IT IS generally agreed that the proper preparation of a patient for roentgen examination of the colon requires the use of both an efficient cathartic and cleansing enemas. For this purpose the most frequently advocated cathartics have been castor oil, compound licorice powder and phenolphthalein. While these are usually used in combination with a pre-examination cleansing enema, some radiologists have relied solely on either laxatives or enemas.

We have been using an agent (X-PREP†), the active ingredient of which is senna, as the laxative component in our preparation for both routine barium enemas and double contrast studies of the colon. We found X-PREP to be most effective in these situations, and a comparative study of its usefulness was undertaken. Since castor oil has been employed most extensively[2,5] as a pre-examination cathartic, it was felt that it should serve as a basis for comparison with X-PREP.

X-PREP consists of a measured amount of cocoa-flavored powder containing the total active constituent of Cassia acutifolia pod (senna), but devoid of the irritating resins of its leaves or seeds. This vegetable substance has been uniformly standardized.

While it had been thought that the action of the senna anthracene compounds was directly in the large bowel mucosa, it has been shown by Triende and Okada in 1937 and by Abrahams[1] in 1940 that absorption occurs in the small bowel and the active principles reach the colon through the blood stream. Presumably, there, enzymes from B. coli act on the senna glycosides and produce new compounds which have a stimulating effect on the neuromuscular (Auerbach's plexus) apparatus. For the most palatable administration, the contents of one container are dissolved by mixing with a spoon in about one glass of milk. The material is thus taken as a single dose the evening before the contemplated roentgen examination.

One hundred consecutive patients (omitting only those where it was contraindicated), who were given either routine barium enemas or double contrast colon studies, form the basis for this report. Each of these patients was questioned on the morning of the examination regarding his reaction to the preparation. In addition, observations were made relative to the quality of the roentgenograms obtained, particularly with regard to the cleansing effect of the laxative administered. In preparing for routine barium enemas, our patients were instructed to eat a low residue meal on the evening prior to the examination; breakfast was omitted and a small saline enema was administered before reporting for roentgen examination. Patients who were to have a double contrast study were instructed to include three cleansing enemas prior to the actual examination.

All of the patients found the material to be palatable and easily prepared. Compared to castor oil, there were fewer complaints of gripping, sleeplessness, and cramping. While about half of the patients were awakened during the night because of an urgent bowel movement, they did not find this particularly objectionable. About 20 per cent of the patients complained of severe cramps lasting from thirty to sixty minutes and occurring approximately eight hours following ingestion of the laxative. These complaints did not impress us as being of a serious nature. By comparison, this type of complaint is present in a higher percentage of patients being

* From the Department of Radiology, Boston University School of Medicine and Massachusetts Memorial Hospitals, Boston, Massachusetts.

† X-PREP supplied by Gray Pharmaceutical Co., Newton, Massachusetts.

given castor oil. During the actual examinations there was not found to be any significant degree of spasm or irritability of the colon. In many cases there was noted to be better cleansing of the distal colon than had been our experience with castor oil. This method of bowel cleansing has been most rewarding in our patients undergoing double contrast enemas. Fictitious polyps of the colon seen on the double contrast study have proved to be a constant source of difficulty for the radiologist.[6,7] Thorough cleansing is the first requisite if these annoying artefacts are to be eliminated. A minor though recognized cause of fictitious polyps is undigested castor oil droplets that appear in the colon following its use as a purgative. This potential source of difficulty can be eliminated by using a non-oily cathartic. The use of X-PREP for our method of double contrast examination of the colon[3] has yielded consistently satisfactory results.

It is hardly necessary to stress that the injudicious use of purgatives is not good medical practice. The usual contraindications to the use of this type of preparation are severe diarrhea, severe intestinal hemorrhage, and acute or subacute obstruction.[4] A cathartic for the preparation of the colon should be used only after a history has been obtained that does not contraindicate its use. We therefore recommend that these conditions be excluded before X-PREP is prescribed. This, of course, applies equally to all cathartics.

SUMMARY

In an attempt to secure the best possible preparation of our patients as well as their willing cooperation prior to roentgen examination of the colon, we have been using X-PREP, which is a palatable senna cathartic. X-PREP is well accepted by the patient and effective as a pre-roentgen examination cleansing cathartic comparable to castor oil and in some respects superior to it for this purpose.

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Fortieth Annual Meeting: Hollywood Beach Hotel, Hollywood Beach, Florida, March 27–29, 1958.

EDITORIALS

PUBLIC RELATIONS AND EDUCATIONAL PROGRAMS OF THE AMERICAN COLLEGE OF RADIOLOGY*

THE public relations and educational programs of the American College of Radiology are based on the desirable standards of radiologic practice as formulated by the College. These concepts were developed by the Board of Chancellors and are not those of the individual members of the Commission on Public Relations. The Commission tries to ascertain the consensus of opinion of the membership, to keep in accord with it at all times, and to express and interpret it to the public.

In the beginning, the Commission had to collect these concepts, ideas and ideals of radiologic practice; bring them together, and organize them into a plan from which public relations and educational programs could be launched. In doing this, it had to present the best type of radiologic practice and the direction in which radiology must go if it is to continue in advancing medical care. The Commission also had to determine the manner in which the radiologist wished to be presented to the public, by what means, and the proper way to tell about his contributions to their medical welfare.

These factors are in a constant state of flux and are in an evolutionary process. The Commission, therefore, keeps in close touch with these developments through the Board of Chancellors and the meetings of the Councillors. The information obtained from these sources can then be translated and transmitted to the public. This is why the Commission has encouraged the Councillors to take an active part in their programs—not only as a means of disseminating information, but also as an important source of news and ideas. The Councillors also serve as a sounding board for the trial and seasoning of new projects.

Public Relations. Public relations can be defined as the everyday manner in which every radiologist and his technicians, secretaries, accountants, nurses and orderlies meet and handle their patients. It is the way in which the patients are greeted, conducted to and from the diagnostic and therapy rooms, and the finesse and skill with which the examinations and treatments are performed. It is consistent courtesy, kindness, tact and efficiency of the entire personnel in our office or department in the hospital. Each day it must grow or shrivel. It is a living and ever changing thing, yet its course can be guided and influence expanded by our daily actions.

It is the accessibility and convenience with which we provide consultations for our medical colleagues; the efficiency with which we accept and complete requested examinations; the dispatch with which our reports are prepared and delivered; the interest and pleasure with which we examine and treat our patients. In fact, public relations is how well we do our professional job and how well we handle our patients. To quote my professor, Dr. Sherwood Moore, "The radiologist is just as good as the referring physicians and patients think he is." These are the reasons why the radiologist must see and interview each patient, for how can the patient or referring physician form an opinion about the "little radiologist who isn't there?" Not even gigantic efforts

* By Invitation, Presented at the Fifty-seventh Annual Meeting of the American Roentgen Ray Society, Los Angeles, California, September 25–28, 1956.

by the experts on Madison Avenue can improve public relations when the object of their endeavor is not available to be seen by those whom he seeks to influence.

It is self-evident then that if the radiologist is to up-grade his public relations he must do it himself—on an individual basis —by constantly cultivating and nurturing his relations with the patients and with the referring physicians. The radiologist becomes his own master in public relations as soon as he comes to know his patients and his doctors.

The task of the Commission in this field is to emphasize and to point out the best principles of radiologic practice. This, at present, is accomplished most by preceptorship training, by articles in the News Letter, by the motion picture, "First a Physician," and by round table discussions at the annual meetings of the College.

Public relations is not the number of booklets, pamphlets, articles, radio or television broadcasts or lectures produced about radiology. They are part of another related effort—the organized program of education for the public about radiology. In this field we need the advice, help and assistance of experts and cooperation with other groups having similar interests.

Educational Programs. In the development of Educational Programs for the public, the Commission has adhered to three basic premises: First, the radiologist is a physician; second, the radiologist is an important member of the modern medical team; third, diagnostic radiologic examinations are clinical examinations and are not technical laboratory procedures.

In emphasizing the first premise, "the radiologist is a physician," we have described his education in college and in the medical school, his residency training in the specialty of radiology, his certification by the American Board of Radiology, his election to the American College of Radiology and how he carries on his practice. This story has been told in our pamphlet, "X-rays Protect You," of which 130,000 copies have been distributed. The pamphlet has now been revised with the suggestions and criticisms that were received. The new edition is more attractive and contains about the same information with minor additions and deletions.

A story about the radiologist was exceptionally well done by Dr. J. D. Ratcliff in his article "What the Doctor Sees In You." This appeared in the October, 1955 issue of *Reader's Digest* which has a circulation of over 10,361,531. About 100,000 reprints of this article were distributed to the members for their reception rooms. This article actually was inspired by the pamphlet, "X-rays Protect You" which shows how important it is to prepare and distribute our own material. The life history of the radiologist was again repeated in the splendid article by Mr. Greer Williams on Dr. Merrill Sosman that appeared in the November 12, 1955 issue of *The Saturday Evening Post* and which reached 4,764,000 readers.

The latest project which tells about the radiologist is the motion picture, "First a Physician" sponsored by the Du Pont Photo Products Division in close cooperation with the Commission and produced by Audio Productions in New York City. The Du Pont Company was agreeable to suggestions concerning the script and to the technical assistance rendered by Dr. Ted Leigh, who stayed in New York throughout the two weeks required for "shooting" the film. It will undoubtedly be shown to many groups of interested people and over television networks to millions of viewers.

The second premise, "the radiologist is an important member of the modern medical team," describes his working relationship with other medical specialists and the place he occupies in determining the diagnosis and treatment of patients. While this premise is also brought out in the material just described, it has been highlighted by the advertisements of the X-ray Department of the General Electric Company published monthly in *Newsweek* with over 900,000 readers. These advertisements carry a strong radiologic impact emphasiz-

ing our position on the modern medical team. All of us have received bound copies of these for our reception rooms. The pictures and script are submitted prior to publication for suggestions and approval by the Commission.

The third premise on which the Commission operates, "diagnostic radiologic examinations are clinical examinations and are not technical laboratory procedures," is probably the most important one the Commission has been called upon to emphasize. It is necessary that the radiologist see and examine the patients and really perform a clinical service. In our relationship with hospitals, state and federal health agencies, it has become vital that radiologic practice be clearly recognized as the clinical practice of medicine. In no way can this be more forcibly brought to the attention of everyone, including the public, hospital administrators, officials of health agencies, legislators and the medical profession, than to be certain every patient is seen by a radiologist or a member of his resident staff. The patient must have the experience of meeting the radiologist who introduces himself as a doctor and makes the patient conscious of the fact he has had a medical examination and not a technical examination performed by a technician. When this is done, there will be no confusion in the minds of the patient as to who is the radiologist and who is the technician. This is the only way in which radiology can be "sold" as a clinical examination. We must act like doctors, look like doctors and talk like doctors if we want to be recognized as doctors.

This principle also applies to the bills sent to patients on which the identity of the radiologist should be easy to recognize —otherwise, he is relegated to the position of a nonentity. In other words, our statements should look like the statements of other doctors.

To see the patients will require more radiologists for the same diagnostic load. This in turn means an increase in the budget for professional personnel with a subsequent narrowing and the elimination of excess funds earned by some departments. It is this excess fund, when it occurs, that focuses the attention of administrators on radiology. To state it simply, our goal should be more and better individual professional radiologic care for the patient at the expense of the profit factor without increasing the cost to the patient. In other words, it looks as if only by increasing professional radiologic care to the patient can we advance radiology and render greater service.

Radiology is a medical service and should occupy the same position within the hospital as other major clinical services.

To further develop the third premise, that radiology is the clinical practice of medicine, the Board of Chancellors authorized the Commission to prepare a quarterly magazine for the purpose of disseminating radiologic information to hospital trustees and administrators. Mr. Hugh Jones, Director of Public Relations, had this idea and it has become "Your Radiologist." This magazine has been a successful endeavor.

The three basic premises are also being reiterated in newspaper articles, press releases, radio and television scripts prepared by the J. Walter Thompson Company for the excellent educational program the Eastman Kodak Company has been carrying on in medicine and particularly in radiology. It is a difficult task to supply articles for newspapers, industrial magazines, trade journals and Sunday magazine supplements and requires a specialized organization with know-how to utilize the complex machinery of communication. The Thompson Company is doing this for the Eastman Kodak Company in cooperation with the Commission. The Eastman Kodak Company has shown a keen desire to carry out the policies and wishes of the College.

The College and the Eastman Kodak Company, as a joint undertaking, have made three four minute movies for television distribution that show comparisons between plant growth and the roentgeno-

graphic demonstration of certain pathologic disorders in the human body by means of time lapse photography. These films have been released, first through the American Medical Association to medical societies, and are now available to all television stations. The films feature Dr. Earl Barth as the radiologist and Mr. John Ott as the gardener.

The annual meetings of the national radiologic societies provide an unexcelled opportunity for telling the public about radiology. Mr. Hugh Jones and his staff prepare "press kits" of information for each meeting. They include abstracts of the papers to be presented, descriptions of the scientific exhibits, a copy of the program and summaries for radio and television scripts. About 279 such "press kits" were mailed to medical and science writers prior to the meeting last September. Mr. Jones is "loaned" to the society for the duration of a meeting and works under the direction of that society's Committee on Public Relations. By this arrangement, the meetings of the scientific societies are now covered by a public relations expert with a radiologic background. For the first time the Societies are enjoying an adequate press with satisfactory television and radio coverage.

Television and major radio broadcasts made in the past year included: The National Broadcasting Company's television program, the "March of Medicine" produced by Smith, Kline and French Laboratories in cooperation with the American Medical Association. It featured an hour-long report on radiology from the September, 1955 meeting of the American Roentgen Ray Society.

The National Broadcasting Company's radio program, "Monitor," devoted twenty minutes to diagnostic and therapeutic radiology during the same meeting.

The Mutual Broadcasting System presented on its "Northwestern Reviewing Stand" a thirty minute discussion on radiation protection at the December meeting of The Radiological Society of North America.

In connection with the sixtieth anniversary of Röntgen's discovery of x-ray, the NBC-TV program called "Today," with Dave Garroway, devoted portions of 3 broadcasts to recent advances in radiology.

In summary, the Commission on Public Relations of the American College of Radiology with the support of the Board of Chancellors and the Councillors working with Mr. Hugh Jones, Director of Public Relations, and with Mr. William Stronach, Executive Director, has developed a sound public relations and educational program for the public. They have been generously assisted and aided by the Eastman Kodak Company, the X-ray Division of the General Electric Company, and the Photo Products Division of Du Pont Chemical Company. The program operates at a high level of activity and makes every effort to portray the radiologist and his specialty in an honest, accurate and dignified manner. I want to close with a word of caution —in telling about ourselves, let us each and every one see that we actually do what we say we are doing; otherwise, all is a sham.

We represent a young specialty in medicine—young enough to hold great promise for the future, vigorous enough to carry a heavy work load, and enthusiastic enough to render better service to our patients through continued research and the improvement of individual professional care.

Wendell G. Scott, M.D.
Chairman, Commission on Public Relations
American College of Radiology

JOSEPH M. FOLEY
1899-1957

ON APRIL 29, 1957, Dr. Joseph Milton Foley, Associate Radiologist at The Grace Hospital, Detroit, Michigan, died suddenly of a recurrent heart attack. A few minutes before his death he had reported for work at the Northwestern Branch. For many months Dr. Foley had been confined either to a hospital bed or at home as the result of a severe coronary thrombosis in August, 1955. Recently he returned to a limited work schedule at Grace Hospital and his sudden passing was a shock to his many friends and admirers.

Dr. Foley started his Detroit radiological practice in April, 1944. He was born in Chicago, March 31, 1899—the only child

of Irish immigrant parents. Early in his boyhood he was sent to St. Joseph's College, Collegeville, Indiana, to complete his preparatory schooling. In order to provide funds for a college and medical education he became an auditor and accountant, and was employed at the Haynes Auto Company, Kokomo, Indiana from 1918 to 1922 as Chief Auditor.

Dr. Foley was an alumnus of the St. Louis University, receiving his Bachelor's Degree in 1922 and his Medical Degree in 1926. He interned at Mercy Hospital, Toledo, Ohio and upon completion of his interneship entered private practice in Toledo. Having decided upon a specialty, he returned to Chicago, the city of his birth, and received his radiological training at St. Luke's Hospital under Dr. E. L. Jenkinson, with whom he continued his association for four additional years. While at St. Luke's and in collaboration with Dr. Jenkinson, Dr. Foley contributed two articles for the American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.

In 1936 he became Director of the X-Ray Department at St. Francis Hospital, Peoria, Illinois, and it was there he met the charming Virginia Merkle, whom he married on October 17, 1938.

That same year Dr. Foley became Director of the Department of Radiology at Wheeling Hospital, Wheeling, West Virginia, and remained there until 1943. Because of frequent sinus attacks while living in West Virginia he decided to return to the Great Lakes area where he had enjoyed good health in his early medical career, and in April, 1944, he became associated with Drs. Stevens, Jarre and Hasley, Radiologists, Detroit, Michigan.

Dr. Foley was a member of the Wayne County Medical Society, the Detroit Roentgen Ray and Radium Society, the American Medical Association, the American Roentgen Ray Society and a Fellow of the American College of Radiology. "Joe" Foley never missed a meeting of the American Roentgen Ray Society by choice. He became active in the organization when he was associated with Dr. Jenkinson and served on some of the important committees. He was known by his confreres as the astute business man, and over the many years he gave unstintingly of his advice and counsel. He was always happy to help—to help in modesty and without acclaim—and to those who knew him he was the unseen and unheralded worker.

His early passing is keenly felt by his colleagues and associates. He is survived by his devoted wife.

Clyde K. Hasley, M.D.

SOCIETY PROCEEDINGS AND NEWS ITEMS*

JOURNAL BELGE DE RADIOLOGIE

It is with pleasure that the American Roentgen Ray Society and the American Radium Society in their official publication salute the *Journal Belge de Radiologie* on the Fiftieth Anniversary of its publication and bespeak for it a continuation of its splendid contribution to radiology. May the years in the future be as distinguished in its accomplishments as the past has been.

AMERICAN BOARD OF RADIOLOGY
Training Requirements

For Candidates Applying In:

RADIOLOGY:

Those beginning their training *before* July 1, 1956 must have three years' formal residency training in an approved department of Radiology.

Those beginning their training on July 1, 1956 or thereafter must have three years' formal residency training in an approved department of Radiology plus an additional year of either further training or practice. The three year training period must include the equivalent of six months (total) in Pathology and Isotopes.

Those beginning their training on July 1, 1956 or thereafter will be required to take a written examination in Physics (including Physics of Isotopes) and Pathology at the *completion* of their formal training. Acceptance for the oral examination is contingent upon passing the written examination. (The written examination in the above two mentioned phases of Radiology will abolish the oral examination in these two phases.)

DIAGNOSTIC ROENTGENOLOGY:

Those beginning their training *before* July 1, 1956 must have two and one-half years' formal residency training in an approved department of Radiology. The equivalent of six months of this time must have been devoted to Pathology.

Those beginning their training on July 1, 1956 must have two and one-half years' formal residency training in an approved department of Radiology (the equivalent of six months of which time must have been devoted to Pathology) plus an additional year of either further training or practice.

Those beginning their training on July 1, 1956 or thereafter will be required to take a written examination in Physics and Pathology at the *completion* of their formal training. Acceptance for the oral examination is contingent upon passing the written examination. (The written examination in the above two mentioned phases of Diagnostic Roentgenology will abolish the oral examination in these two phases.)

Those beginning their training on July 1, 1957 or thereafter will be required to take three years' formal residency training in an approved department of Radiology —the first year of which must be spent in basic training (Pathology, Physics and Basic Radiologic Techniques) followed by two years' clinical training. They will also be required to take an additional year of either further training or practice. (Written examination also required for these candidates. See above paragraph.)

THERAPEUTIC RADIOLOGY:

Those beginning their training *before* July 1, 1956 must have two and one-half years' formal residency training in an approved department of Radiology. The equivalent of six months of this time must have been devoted to Pathology.

Those beginning their training on July 1, 1956 must have two and one-half years' formal residency training in an approved department of Radiology (the equivalent

* This list of Meetings of the Radiological Societies appears in the July, 1957, issue of the Journal, and will be published in the November number.

of six months of which time must have been devoted to Pathology) plus an additional year of either further training or practice.

Those beginning their training on July 1, 1956 or thereafter will be required to take a written examination in Physics (including Physics of Isotopes) and Pathology at the *completion* of their formal training. Acceptance for the oral examination is contingent upon passing the written examination. (The written examination in the above two mentioned phases of Therapeutic Radiology will abolish the oral examination in these two phases.)

Those beginning their training on July 1, 1957 or thereafter will be required to take three years' formal residency training in an approved department of Radiology—the first year of which must be spent in basic training (Pathology, Physics, Physics of Isotopes and Basic Radiologic Techniques) followed by two years' clinical training. They will also be required to take an additional year of either further training or practice. (Written examination also required for these candidates. See above paragraph.)

Those applying for Therapeutic Radiology alone must show evidence of personal experience in the use of intracavitary and interstitial radium or equivalent sources, as well as proficiency in roentgen therapy.

Prior to July 1, 1960, those candidates desiring to be certified in either Radiology or Therapeutic Radiology, will be urged to take an examination in Nuclear Medicine. After July 1, 1960, this examination will be mandatory.

Those candidates previously certified in Roentgenology desiring to complete their certificate in Radiology (including Nuclear Medicine) will be required to have the equivalent of six months' full time radium and isotope training in an approved department of Radiology.

Prior to July 1, 1960, those candidates who have been previously certified in Roentgenology and who *do not* desire to be examined in Nuclear Medicine must have the equivalent of three months' full time radium training in an approved department of Radiology.

CENTRE ANTOINE BÉCLÈRE

The general assembly of the Centre Antoine Béclère (7 rue Perronet, Paris 7e) on June 13, 1957, has unanimously ratified the nomination by the council on administration of Professor Antoine Lacassagne, Honorary Director of Laboratoire Pasteur de l'Institut du Radium, as president of the Center, succeeding Professor A. Baudouin, recently deceased.

COURSE IN RADIOACTIVE ISOTOPES FOR TECHNICIANS

QUEENS HOSPITAL CENTER

A course to be devoted to laboratory techniques in the use of radioactive isotopes will be given for Technicians. The course, which is limited to 10 members, will consist of 8 sessions of 4 hours each, commencing Friday, October 18, 1957 (1:00 to 5:00 P.M.). Tuition: $100.

Requests for applications should be sent to Dr. Philip Kahan, Supervising Medical Superintendent, Queens Hospital Center, 82–68 164th Street, Jamaica 32, New York.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Les Tumeurs de l'Amygdale et de la Région Vélopalatine. By A. Ennuyer and J.-P. Bataini, Médecins de la Fondation Curie. Paper. Price, 5,000 fr. Pp. 536, with 150 figures and 91 tables. Masson et Cie, Éditeurs, 120 boulevard Saint-Germain, Paris 6e, France, 1956.

This is a thorough study of the tumors arising in the tonsil and soft palate, based on an analysis of cases seen and treated at the Radium Institute of the University of Paris over a period of nearly 40 years. The material is analyzed from a clinical, histopathologic and therapeutic point of view. A great deal of space is devoted to the techniques of treatment. In the treatment of carcinomas of the tonsil the association of external roentgen therapy and interstitial radium therapy has yielded 5 year survivals of nearly 30 per cent. There are many illustrated summaries of histories chosen from among the cured patients.

This valuable monograph is the best study of its kind to appear in print since Berven's monograph, published in 1931.

J. A. del Regato, M.D.

Current Therapy—1957; Latest Approved Methods of Treatment for the Practicing Physician. Edited by Howard F. Conn, M.D. Cloth. Price, $11.00. Pp. 731. W. B. Saunders Company, 218 West Washington Square, Philadelphia 5, Pennsylvania, 1957.

In this ninth annual volume the editor and his outstanding group of consultants describe the treatment of some 400 common disease states, devoting more than a third of the book to new and modified forms of treatment. Thus the aim of bringing to the practitioner the latest in therapy is amply fulfilled.

There is much help available in this practical work for the physician seeking competent therapeutic guidance after a diagnosis has been established.

William S. Reveno, M.D.

Pelvimetry. By Herbert Thoms, M.D., Emeritus Professor of Obstetrics and Gynecology, Yale University School of Medicine, New Haven, Connecticut. Cloth. Price, $5.00. Pp. 120, with 45 illustrations. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1956.

This monograph is primarily a survey of the numerous papers which have been published by the author and his associates during the past 25 years. It presents a description of the variations and abnormalities of the bony pelvis in relation to labor and the roentgen and clinical diagnostic procedures by which pelvic contour and capacity can be ascertained.

In a sense the text is very limited in scope, omitting all but casual reference to the fetus, soft tissues or clinical obstetrics. The roentgen examination is also minimal, consisting of two roentgenograms of the pelvis, one obtained by projection from above downward with the target centered on a line at right angles to the plane of the pelvic inlet, and the other projected laterally. By this technique the author determines the shape and dimensions of the pelvic inlet, and the measurement of the midplane, while in the lateral view the lateral aspect of the pelvis is studied and certain anteroposterior dimensions obtained. Certain outlet characteristics can be shown but the author believes the outlet can be satisfactorily evaluated by palpatory methods.

Since this text is a review chiefly of the author's own experience and previous publications the concept presented here is not new, but it is easily understood and practical to use.

Karl K. Latteier, M.D.

Studio Clinico-Radiologico Degli Esiti dell'Ascesso Polmonare. By Francesco Santagati, Nardo Calvi, and Giancarlo Rivolta. Paper. Pp. 56, with 88 illustrations. Ind. Graf. Carlo Re & C., Via Neera 50, Milan, Italy, 1956.

The authors have had the opportunity of studying the chests of inmates of a mental hospital, and a number of these patients had old healed or dormant lung abscesses.

These patients were meticulously examined with plain films, laminagrams and bronchograms, and it was found that even in those patients who were clinically well there were per-

manent alterations of the lungs, of the pleurae and of the bronchial tree.

This monograph cannot be appreciated unless one sees the many beautiful illustrations. The bronchograms are especially striking and show an exceptional mastery of the technique of bronchography. Forty-eight cases of old lung abscesses are thus described with adequate clinical data and excellent roentgenographic studies.

Cesare Gianturco, M.D.

Diseases of the Skin. By George Clinton Andrews, M.D., F.A.C.P., Clinical Professor of Dermatology, the College of Physicians and Surgeons, Columbia University; Attending Dermatologist to the Presbyterian Hospital, Columbia-Presbyterian Medical Center, New York. Fourth edition. Cloth. Price, $13.00. Pp. 877, with 777 illustrations. W. B. Saunders Company, 218 West Washington Square, Philadelphia 5, Pennsylvania, 1954.

This is the fourth revision of an excellent reference text in this field. Its wide usage is exemplified by its presence in the libraries of the U. S. Navy, on sea aboard ship and ashore. Its great value undoubtedly stems from its transportable size (a quality often lacking in modern texts of encyclopedic scope) as well as its complete and succinct treatment of skin diseases.

This edition differs from its predecessor of eight years in its modern treatment of countless disorders. There is increased emphasis on histopathology of the skin. The chapter on tumors has been revised. There has been expansion and revision on the viral and fungous diseases as well as modernization of the "collagen disease group." Countless new syndromes and diseases have been added.

Of the features which distinguish this volume, treatment is perhaps foremost. A volume such as this is of value when used with sufficient background and practical experience. Since therapy is presented in detail, and includes many empiric remedies of questionable value, the average physician, in selecting a treatment for a skin disease listed, may apply a medication as his first treatment which is best reserved for situations of more chronic or severe nature, with a resulting overtreatment dermatitis. For the dermatologist this detailed reference to therapy is helpful, but it is of less value to the general practitioner.

There is a very good section, consisting of three chapters, on the physics and practical application of the roentgen rays and radium. The discussion of roentgen-ray physics in dermatology is masterfully presented by Carl B. Braestrup. This material expertly summarizes the common practical application of these modalities.

This volume should be on the bookshelf of anyone interested in the diagnosis and treatment of diseases of the skin and can be fully recommended.

Arthur C. Curtis, M.D.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Blatt, Nicolas, and Athanasiu, Mircea. (Bucarest, Romania.) Étude radiologique des corrélations entre le canal optique et le sinus sphénoïdal. (Roentgenologic study of correlations between the optic canal and the sphenoidal sinus.) *J. de radiol., d'électrol. et de méd. nucléaire*, March-April, 1957, *38*, 158–168.

Two hundred roentgenograms were studied from the anatomic and roentgenologic point of view. The authors found the sphenoidal sinus of very large dimensions in 20 per cent of the cases, of average dimensions in 30 per cent, small in 37.5 per cent and very small in 12.5 per cent. The fact is that all small sinuses, having minimal neighborhood relations with the optic canal, will only rarely reach the optic canal giving rise to an optic neuritis.

They found that only 20 per cent of the cases of sphenoidal sinusitis will predispose to optic neuritis because of the large dimensions of the sinuses. *Maurice Dufresne, M.D.*

Owen, Morris. A device for the localization of dental remnants in edentulous regions. *Radiology*, April, 1947, *68*, 582–583. (Address: Royal Newcastle Hospital, Newcastle, New South Wales, Australia.)

The author describes a simple, inexpensive device for facilitating the localization of dental remnants in edentulous regions of the alveolar margins. It consists of a lead ruler (constructed from the backing of commerical occlusal film packets) which has a serrated edge with the teeth being at 0.5 cm. intervals and a hole in the center at 2.5 cm. from either end. The ruler is fixed to the tube side of the dental film with cellulose adhesive tape and the roentgen exposure is made in the usual way with either end of the ruler aligned with the midpoint of the alveolus. The processed film will show the remnant and also the image of the ruler, which indicates the distance of the remnant from the midline of the alveolar margin.—*Donald N. Dysart, M.D.*

Rubin, Philip, and Besse, Byron E., Jr. The sialographic differentiation of Mikulicz's disease and Mikulicz's syndrome. *Radiology*, April, 1957, *68*, 477–487. (Adress: P. Rubin, Radiation Branch, National Cancer Institute, Bethesda 14, Md.)

The authors discuss the clinical, pathologic, and roentgenographic aspects of Mikulicz's disease and Mikulicz's syndrome, correlating the pathologic and roentgen features of these conditions. They present excellent diagrams and roentgenographic reproductions illustrating the pertinent points of their study.

In Mikulicz's disease, the main roentgenographic feature is sialectasis, which is divided into four stages: punctate, globular, cavitary and destructive. Briefly, there occurs a progressive narrowing of the second to fourth order ducts to eventual obliteration. As this process develops, the peripheral ducts dilate progressively, giving the above mentioned stages on the sialogram. In the fourth or destructive stage, no recognizable branching duct system in the gland is found sialographically. The radiopaque material dissects its way into the septa of the gland and the subscapular space.

In Mikulicz's syndrome, the duct system remains intact until the pathologic process passes from the surrounding lymph nodes into the gland, destroying the duct system in this late phase. There is progressive outward displacement of the duct system while the lymph nodes are enlarging. There is good emptying of the gland until invasion occurs.

In the end stages of both Mikulicz's disease and syndrome, the gland architecture is completely replaced or destroyed; in the former, the radiopaque medium can be injected readily, whereas in the latter, injection is impossible because the main duct is obstructed.—*Dean Nichols, M.D.*

Neck and Chest

Nagle, W. William, Hope, John W., and Bongiovanni, Alfred M. Congenital goiter. *Radiology*, April, 1957, *68*, 526–533. (Address: J. W. Hope, 1740 Bainbridge St., Philadelphia 46, Pa.)

Congenital goiter occurs rarely in the United States and the authors report 3 cases seen by them in three years. A discussion of the roentgen and clinical findings, pathology, etiology and treatment is presented, along with a review of 70 cases reported in the English literature.

The patient is usually born with a mass in the thyroid region. The mass is soft and symmetric. Symptoms vary but most commonly consist of respiratory distress, the severity of which depends on the size and location of the mass. Roentgenograms, which should be taken in full inspiration, generally show an evenly outlined soft-tissue mass in the thyroid region that surrounds the trachea, and sometimes the esophagus, compressing and displacing these structures. These three cases, and the vast majority of other infants born with thyroid enlargement, show no evidence of thyroid hormone abnormality.

The pathologic findings in most of the 25 pathologic studies available consisted of hyperplastic changes with occasional involutional changes being present. The etiology is not known even though most infants with congenital goiter have been born to mothers with evidence of thyroid disease. Factors considered are maternal thyroid disease and the in-

gestion by the mother of goitrogenic drugs, the goitrogenic action of calcium in low iodine areas, and the geographic "goiter belts."

The treatment consists essentially of maintenance of an adequate airway. If the thyroid enlargement is most marked posteriorly, tracheotomy may be necessary and it may be that a subtotal thyroidectomy will be advisable. Usually the enlargement will diminish in a few weeks. If thyrotoxic symptoms are present the administration of iodides either by mouth or parenterally will rapidly and effectively decrease the size.—*Donald N. Dysart, M.D.*

Gombert, H.-J., and Kloppe, W. Zur Diagnose des metastasierenden Schilddrüsenadenoms. (The diagnosis of metastasizing thyroid adenoma.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May, 1957, *86*, 567–575. (Address: H.-J. Gombert, Städt. Krankenhaus, Lynarstr. 12, Berlin-Spandau, Germany.)

Metastasizing adenoma of the thyroid represents a rare entity, constituting 2.4 per cent of all thyroid tumors. The authors have collected only 4 cases in a twenty year period. This condition has been identified as "angio-invasive adenoma" and "carcinoma adenomatosum microfolliculare" by other authors.

The diagnosis can be made only by pathologic study, since no clinical, roentgenologic, or radioactive-iodine uptake characteristics are of value. The metastatic lesions are generally highly vascular, frequently pulsatile on palpation, soft and spongy, slowly growing, osteolytic, and probably never osteoblastic.—*Joseph Zausner, M.D.*

Mazzei, Egidio S., and Masnatta, Guillermo. Cuadros pleuropulmonares en la leucemia aguda (pulmon leucemico agudo). (Pleural pulmonary picture in acute leukemia [acute pulmonary leukemia].) *Prensa méd. argent.*, Jan., 1957, *44*, 1–5. (Address: E. S. Mazzei, Hospital de Clinicas de Buenos Aires, Buenos Aires, Argentina.)

Four cases of acute leukemia with pulmonary manifestations are reported. *Case 1:* Acute lymphatic leukemia with a hemothorax on the right side at the onset of the disease; *Case 2:* Acute monocytic leukemia with evidence of pulmonary disease on the right side; *Case 3:* Acute myeloid leukemia with a right sided pleurisy; *Case 4:* Acute hemocytoblastic leukemia with roentgen ray evidence of disease on the left side.

The underlying pathologic changes in the lungs are caused by parenchymal infiltration beginning in the region of the alveolar septum. Subsequently there may be bronchial or bronchiolar compression, subpleural infiltration or infiltration contiguous to the blood vessels. Roentgenologically, the pulmonary changes are similar to changes caused by other pulmonary affections such as tuberculosis, tumor, pleurisy and other acute inflammatory disease. These conditions must be ruled out before a diagnosis of leukemic infiltration can be made.—*Murray M. Friedman, M.D.*

Hankamp, LaMar J. Calcification of the myocardium; case report. *Radiology*, April, 1957, *68*, 564–567. (Address: St. Joseph Mercy Hospital, Ann Arbor, Mich.)

The author presents a case report of a patient who exhibited calcification of the myocardium which was evident on the chest roentgenogram. Although the history did not indicate any single episode which would suggest a myocardial infarction, the electrocardiogram showed the presence of an old anteroseptal and lateral left ventricular infarction.

Roentgenograms of the chest showed a curvilinear calcific deposit related to the apex of the heart about 1 cm. medial to the left cardiac border. Fluoroscopically this was seen to move synchronously with ventricular contractions, and appeared to be located in the region of the interventricular septum. Heart size and shape were normal.

The differential diagnosis includes calcification of valves or mitral annulus, coronary artery calcification, calcification of the left atrial endocardium or mural thrombus calcification associated with mitral stenosis, and pericardial calcification. A brief discussion of the pathologic basis for cardiac calcification is presented.—*Donald N. Dysart, M.D.*

Moore, Thomas C., Battersby, J. Stanley, Roggenkamp, Milton W., and Campbell, J. A. Congenital posterolateral diaphragmatic hernia in the newborn. *Surg., Gynec. & Obst.*, June, 1957, *104*, 675–689. (From: Departments of Surgery, Pathology, and Radiology, Indiana University Medical Center, Indianapolis, and the Surgical Service, Ball Memorial Hospital, Muncie, Ind.)

Sixteen cases of congenital diaphragmatic hernia in the newborn are reported. Thirteen of these cases were diagnosed clinically and managed by operation while the diagnosis was made on a postmortem examination in 3 cases.

The diagnostic triad of dyspnea, cyanosis and "dextrocardia" was observed consistently in those infants in whom the diagnosis was made during the early hours of life. The frequent occurrence in these infants of the illusion of dextrocardia, secondary to the shift of the mediastinum into the right hemithorax, is emphasized.

All of the patients had left diaphragmatic hernias, which were basically of the posterolateral, foramen of Bochdalek type. A hernia sac occurred in 3 of the 16 cases. The likelihood of not discovering the pres-

ence of a hernia sac in infants operated upon through an abdominal incision during the early hours of life is discussed. Presumably this failure to identify a sac, when present, results from its close approximation by the negative intrathoracic pressure to the collapsed and nonexpansible lung. Closure of the hernia defect in the presence of an unrecognized sac creates a large cyst on the superior surface of the involved diaphragm. In order to avoid this complication when an abdominal incision is employed, the supplemental use either of a small intercostal thoracic incision or an incision in the pleura of the chest wall from the inside to release the negative intrathoracic pressure and permit a sac, if present, to fall away from the nonexpansible lung is suggested.

All of the 13 patients who were managed by operation were treated during the first ninety hours of life, 11 of them being under forty hours of age at the time of operation. All older infants were excluded from this study. An abdominal incision was employed in 11 cases and a thoracic incision in 2 cases. Eight infants recovered and 5 died.

At operation, the ipsolateral lung was found in a majority of cases, to be small, collapsed and nonexpansible. No expansion of the left lung could be obtained at the time of operation in 8 of 11 cases. Expansibility was minimal in 2 cases and moderate in 1. Postoperative roentgen studies suggest that, if the infant survives, slow but progressive expansion of the left lung may be expected. This observation was repeatedly made in cases in which the left lung at operation was observed to be small, collapsed, and nonexpansible. Both the futility and the hazard of vigorous attempts to expand the small, collapsed, and nonexpansible left lung by positive pressure through an endotracheal tube at operation or by chest suction immediately following operation, are emphasized.

Early operation is recommended to provide an optimum climate for the slow and progressive expansion of the left lung and to avoid increasing pulmonary compression from the distention of intrathoracic gastric and intestinal viscera with swallowed air.—*James F. Martin, M.D.*

Boyd, David P., and Woolridge, Bart F. Diaphragmatic hernia through the foramen of Morgagni. *Surg., Gynec. & Obst.*, June, 1957, *104*, 727–732. (From: Department of Surgery, The Lahey Clinic, Boston, Mass.)

The authors describe their experience in the diagnosis and treatment of 8 patients with diaphragmatic hernia through the foramen of Morgagni.

Morgagni first described herniation through the retrosternal aspect of the diaphragm behind the xiphoid cartilage in a monograph written in the eighteenth century and this aperture has since been known as the foramen of Morgagni. The diaphragm originates essentially from 4 areas—sternal, costal, crural and the arcuate ligaments. The sternal origin consists of short right and left slips from the posterior aspect of the xiphoid process which are separated by areolar tissue and insert into the anterior margin of the central tendon. A triangular space, devoid of muscle fibers, is between the sternal and anterior costal attachments in the diaphragm. It is covered above by the pleura and pericardium and below by the peritoneum.

Cardio-respiratory symptoms are said to be the most common, but in this series gastrointestinal complaints predominated. Cough, precordial pain, retrosternal pressure, palpitation and dyspnea are produced by the mechanical presence of the hernia within the chest. Gastrointestinal symptoms may mimic those of disease of the gallbladder, colon, pancreas, stomach or duodenum.

Herniations through the foramen of Morgagni are characterized roentgenographically by a rounded soft tissue density in the cardiophrenic angle with sharp, well-defined borders which, as a rule, cannot be separated from the right border of the heart. The mass does not pulsate and the adjacent lung may be compressed. Fluid levels and gas may be identified within the mass when it contains small intestine or colon. Pneumothorax, pneumoperitoneum and barium studies of the intestinal tract provide additional information. The roentgen diagnosis is not definite if the hernial sac contains only omentum.

Surgical intervention is recommended for the reduction and repair of the hernia and to establish the diagnosis. The transabdominal approach is preferred, with excision of the hernial sac and repair of the defect by various methods.

The authors' series includes 8 patients treated surgically, one of whom had a second operation. The hernia presented on the right side in 8 instances and in 1, a second hernia was identified on the left side two years after repair of a large right-sided hernia through the foramen of Morgagni.—*James F. Martin, M.D.*

Abdomen

Nasio, Juan. Cuerpos extraños del estomago. (Gastric foreign bodies.) *Rev. brasil. de gastroenterol.*, March-April, 1957, *9*, 47–50. (Address: Arenales 1335, Buenos Aires, Argentina.)

Foreign bodies of the stomach can be classified as extrinsic and intrinsic. Extrinsic foreign bodies may originate in the bronchi or biliary tree. Most are swallowed accidentally or deliberately, and are of all sizes, forms and shapes. Sharp pointed objects can cause ulceration perforation of the stomach, with resulting peritonitis. As an aid to roentgenologic diagnosis, sodium bicarbonate can be administered by mouth as a contrast medium. An esophagoscope or a rigid gastroscope can be used to remove a foreign body. When it is not feasible to use this method, the

author has found the immediate administration of boiled or preserved asparagus to be very helpful. Sometimes, it is possible to introduce a magnet into the stomach for removal of a metallic foreign body. In practice, the majority of foreign bodies do not require surgical treatment. More than 50 per cent are evacuated spontaneously, and the remainder, especially in children, are extracted by mouth.

Intrinsic foreign bodies (bezoars) are classified as trichobezoars, phytobezoars and trichophytobezoars or mixtures. The symptoms are not typical. There may be epigastric pain, nausea, vomiting, pyrosis and sometimes halitosis. In general, the symptoms appear at the onset of complication such as gastritis, gastric ulcers, anorexia, polydipsia etc. Generally, symptoms are present for a period of about six months before a bezoar becomes apparent. Bezoars do not cause gastric ulcers, but they tend to aggravate a pre-existing ulcer.

Clinical examination may show a mobile, slightly painful, abdominal tumor. Roentgenologically a filling defect, resembling a tumor, can be seen in the stomach, but it is not fixed.

Bezoars may be complicated by gastritis, ulcers, hematemesis, necrosis, stenosis, diarrhea, intestinal obstruction and ulcerative perforation of the stomach.

In some cases, the gastric stones may be dissolved by means of alkaline lavages and alkaline diet. Breaking up of the concretion by palpating the abdomen must be done very cautiously in order to avoid a perforation of the stomach. Failure of medical treatment and the presence of a complicating organic lesion requires surgical intervention.—*Murray M. Friedman, M.D.*

Johnson, H. Daintree, and Stansfeld, Alfred. Giant rugal hypertrophy of the stomach. *Brit. J. Surg.*, March, 1957, *44*, 517–520. (From: The Departments of Surgery and Pathology, Royal Free Hospital, London, England.)

The authors report a case of giant rugal hypertrophy of the stomach in a forty-eight year-old male who complained of tiredness, breathlessness and presented an anemia. On careful questioning a long-standing history of mild indigestion, relieved by alkalis, was obtained. There was no history of vomiting or melena. There was a definite anemia with hemoglobin of 8 gm. per 100 cc., color index 0.87.

Barium meal studies of the stomach revealed "an odd irregular filling defect on the lesser curvature of the stomach, not typical of a new growth, possibly due to swelling of the mucosa." These findings were confirmed on re-examination, showing in addition destruction of the mucosa and ulceration. These were considered suggestive of neoplasm. Other laboratory tests gave no significant information.

At operation the stomach was moderately enlarged. A soft sausage shaped mass was felt attached to the lesser curvature from the cardia to about 3 inches from the pylorus.

When opened the resected stomach presented enormous rugae; one at the lesser curvature and one on either side, tapering at either end. The appearance was neither that of carcinoma nor of sarcoma. Because of its bulk and friability the hypertrophied mucous membrane made anastomosis with the esophagus difficult. The anastomosis was leaking. The patient took a turn to the worse and died on the seventh postoperative day.

The histologic diagnosis of rugal hypertrophy with no evidence of gastritis or of gastric neoplasm was established.

The literature reveals that this condition has a definite predilection for the greater curvature which makes the authors' case, occurring on the lesser curvature, all the more noteworthy. The etiology remains unknown.

The authors feel that after the roentgen examination, a gastroscopy should have been done and at the operating table the stomach should have been opened before resection. This would have precluded the erroneous diagnosis of malignant disease and the necessity of gastric resection which in this case was not successful.

Ten figures including 3 reproductions of roentgenograms accompany this article.—*William H. Shehadi, M.D.*

Davis, Leonard, and Lowman, Robert M. Roentgen criteria of impending perforation of the cecum. *Radiology*, April, 1957, *68*, 542–548. (Address: R. M. Lowman, Grace-New Haven Community Hospital, 789 Howard Avenue, New Haven 4, Conn.)

Perforation of the cecum is a serious complication of large bowel obstruction. Primary factors in the course of cecal perforation in distal large bowel obstruction are the state of the ileocecal valve and mechanical overdistention. Ischemia, increased virulence of bacterial flora, and biochemical changes occupy more poorly defined roles. The immediate hospital mortality is intimately related to the integrity of the mucosa. The prognosis is best with the mucosa intact, becoming almost hopeless with mucosal disruption. Consequently, it is vitally important to be forewarned of the imminence of cecal perforation. Clinical signs and symptoms are exceedingly variable and do not furnish a reliable basis for diagnosis of impending perforation.

The authors report the results of a study of 7 cases of cecal perforation and 12 cases of impending perforation of the cecum. All patients were studied roentgenographically and the findings were confirmed at surgery or postmortem. Measurements were made of the greatest transverse diameter of the cecum on a prone roentgenogram of the abdomen in all these

cases and in 100 controls without obstruction. Distention was induced artificially in the controls by the injection of air and barium. On the basis of this survey, 9 cm. was established as the critical diameter beyond which danger of perforation exists.—*Arno W. Sommer, M.D.*

Herson, Ronald E. Meconium ileus. *Radiology*, April, 1957, *68*, 568–571. (Address: 622 West 168th St., New York 32, N. Y.)

The author reviewed all cases of meconium ileus encountered at Babies Hospital (New York) from 1945 through 1955 and proved by surgery or necropsy. One case was discarded because adequate preoperative roentgenograms were not available, leaving 17 cases for analysis. Roentgenographic evidence of distal small bowel obstruction was present in all but one case, in which examinations were performed at four and eight hours after birth. Air bubbles were shown in 12 of the 17 patients. Fluid levels were observed in 10 patients.

The usual signs of meconium ileus include progressive distention of the abdomen, vomiting and failure of the infant to pass meconium. Often the diagnosis can be made on the basis of palpation of the meconium mass and roentgen demonstration of air bubbles as described by Neuhauser. Evidence of small bowel obstruction is almost invariably present.—*Dean Nichols, M.D.*

Brockis, J. G., and Gilbert, M. C. Intestinal obstruction by gall-stones; a review of 179 cases. *Brit. J. Surg.*, March, 1957, *44*, 461–466. (From: Cardiff Royal Infirmary, Cardiff, Wales.)

The authors review the literature and present 12 cases of their own. The seriousness of intestinal obstruction caused by gallstones with a high mortality is due to late diagnosis, advanced age of the patient which is further aggravated by dehydration, and electrolyte imbalance due to vomiting.

The incidence of occurrence in the male as compared with the female presents a ratio of 1:7, which compares with the incidence of cholecystitis which is usually 1:8. The most common age is between sixty and eighty years. A progressive decline in mortality is noted due to the fact that patients are being admitted to the hospital earlier during their illness.

The gallstone passes through a fistulous tract between the gallbladder and the intestines, the most common being a cholecystoduodenal fistula, but connections between the gallbladder and jejunum, colon and stomach, though less frequent, do occur.

In 132 cases the site of obstruction occurred in: (1) Ileum, 72 per cent; (2) jejunum, 17 per cent; and (3) distributed btweeen the stomach, duodenum, and colon, 11 per cent.

Although not constantly present or readily detectable, the roentgen criteria for the diagnosis are: (1) Gas in the bile ducts; (2) fluid levels in the intestines; and (3) demonstration of the shadow of a gallstone.

Fluid levels in the intestines constitute the one sign most frequently present.

To prevent the recurrence of the gallstone obstruction, it is essential to carefully explore the patient's abdomen during operation, in search for other calculi which may be elsewhere in the intestines or still in the gallbladder. The latter should be removed if the patient's condition permits, if not at least a cholecystotomy should be done with removal of the calculi. The authors stress this because they had a patient in whom a second gallstone obstruction occurred during the immediate postoperative period. Similar cases have also been reported in the literature.

One table and 3 figures accompany this article. —*William H. Shehadi, M.D.*

Hartman, Albert W., and Hills, William J. (San Antonio, Texas.) Rupture of colon in infants during barium enema; report of two cases. *Ann. Surg.*, May, 1957, *145*, 712–717.

Two cases of rupture of the colon during barium enema examination are presented.

The first patient was a four day old infant boy in whom the barium was seen to spread over the abdominal cavity as soon as the injection was started. Surgical exploration done in two hours after the barium enema, revealed a 3 cm. longitudinal tear in the rectum beginning at the peritoneal reflexion. A transverse colostomy was performed and the abdomen closed without drainage. The patient expired thirty-seven hours after the operation.

The second patient was an eight month old male who was admitted to the hospital after the barium enema had been performed by a technician at a physician's office at approximately 9:30 A.M. on the morning of admission. According to the mother, when the tube was first inserted and the balloon blown up, the child screamed and expelled the tube and inflated balloon, became pale and vomited. He was admitted to the hospital approximately nine hours after the performance of the barium enema. A roentgen examination revealed barium throughout the peritoneal cavity and a large amount of free air beneath the diaphragm. A surgical exploration was performed at which time a large amount of fluid and air rushed out on opening the peritoneal cavity. The fluid contained barium, stool, and exudate. Barium adhered to the bowel, liver and peritoneal surfaces in gum-like plaques. A longitudinal tear was found in the rectum beginning at the peritoneal reflexion and extended proximally for 5 to 7 cm. The cavity was closed and a sigmoid loop colostomy was performed. The patient has recovered and was discharged from the hospital two weeks after admission.

Rupture of the normal colon, by the pressure of the barium enema, seems most unlikely. Most of the

reports in the literature concern rupture of diseased colons. Experimental work demonstrated that the intestine would bear a pressure of 50 to 60 cm. of mercury. All intestines would rupture with pressures over 70 cm. of mercury. Children under twelve years of age supported higher pressures than did adults.

The authors feel that it is logical to assume that rupture occurs from distention of the balloon at a point where the bowel is more or less restricted in its ability to dilate by the peritoneal reflexion, or where dilatation of the colon is limited by its proximity to the peritoneal attachments.

The treatment consists of prompt operation with necessary supportive measures. Wide exposure of the abdominal cavity with careful and adequate cleaning of all the spaces using copious amounts of sterile warm saline is indicated. The rent must be closed by one of the acceptable methods and a proximal colostomy established. The mortality rate is approximately 35 per cent. The cause of death is primarily generalized peritonitis.—*James F. Martin, M.D.*

Wise, Robert E., Johnston, David O., and Salzman, Ferdinand A. The intravenous cholangiographic diagnosis of partial obstruction of the common bile duct. *Radiology*, April, 1957, *68*, 507–525. (Address: R. E. Wise, Lahey Clinic, Boston 15, Mass.)

In an attempt to evaluate the role of intravenous cholangiography in the diagnosis of partial obstruction of the common bile duct, to determine the relationship of common duct size to partial obstructions of the duct, and to establish criteria for roentgenographic diagnosis, the authors have reviewed their series of 770 injections in 746 patients. In 256 patients, the gallbladder was still present and visualization was obtained in 228, while of 490 patients who had had a cholecystectomy, visualization was obtained in 410. Cholografin studies were not done if serum bilirubin values of over 4 mg. per cent were present, if there was a bromsulphalein retention of over 40 per cent, or if clinical jaundice was present. Reactions to the medium were minimal and no fatalities were encountered. The technique of the examination is described.

A study of the common duct sizes suggests that only in the range of diameters of 3 mm. to less than 5 mm. is size alone indicative of completely normal common duct in the postcholecystectomy patient. In the range from 16 to 30 mm., size alone appears to be indicative of some degree of obstruction. Thus, in the range from 5 to 15 mm., size alone is of no value in predicting patency or obstruction. It is in these instances that the time-density-retention relationship over a two-hour period is of benefit. In the completely unobstructed duct, there is less density or retention of medium as seen on the sixty-minute film. Partially obstructed ducts usually show at two hours either the same or greater density than at one hour. This relationship holds only if the gallbladder has been removed or if it does not fill refluxly through the cystic duct.

In the patients with good visualization of both the gallbladder and common duct the duct diameter ranged from 2 to 14 mm. with a presumably normal average of 5.5 mm. In those patients in whom only the common duct was visualized, the size ranged from 3 to 22 mm. with an average of 9.3 mm. Common ducts containing calculi ranged from 4 to 17 mm. in diameter. The authors feel that nonvisualization of the gallbladder in the presence of visualization of the bile ducts is presumptive evidence of a functionless gallbladder due either to a fibrosed lumen or one tightly packed with stones or an obstructed cystic duct.

Common duct calculi were found in 31 patients who had surgery. In only 18 had they been visualized on cholografin studies and in 12 their presence was indicated on the basis of abnormal retention. Calculi that were impacted in the ampulla in general were not visualized as they cannot be detected unless they are at least partially surrounded by medium.

Fibrosis of the sphincter of Oddi and ampulla of Vater was suggested by the finding of partial obstruction as indicated by the time-density-retention concept. Strictures may be diagnosed by the marked difference in size of the duct above and below the point of partial obstruction. Tumors in the ducts or the surrounding structures may, if they are strategically located, cause increased retention and show the presence of a partial obstruction.

This series does not show any evidence to support the concept that there is a postcholecystectomy compensatory dilatation of the common bile duct. The authors feel that many organic causes of postcholecystectomy symptomatology can be diagnosed with the use of cholografin studies, and by excluding these causes the clinician can search elsewhere for the cause of the symptoms knowing that the biliary tract is not at fault.—*Donald N. Dysart, M.D.*

Le Quesne, L. P., and Ranger, I. Cholecystitis glandularis proliferans. *Brit. J. Surg.*, March, 1957, *44*, 447–457. (From: The Department of Surgical Studies and Bland Sutton Institute, The Middlesex Hospital, London, England.)

In this excellent article, the authors review the literature and report 12 cases of their own. Of these, 4 cases were discovered incidentally at autopsy, and 8 cases were encountered in clinical practice.

The outstanding lesion is the formation of epithelial sinuses which penetrate through a muscular layer in the gallbladder wall and expand in the perimuscular or subserous layer, forming cyst-like spaces. The "neck" of these cyst-like spaces may become occluded, giving rise to cysts which roentgenologically appear like filling defects erroneously inter-

preted as a papilloma or tumor. There is an associated muscular hypertrophy, chronic inflammatory changes, and intramural calculus formation.

The authors stress the thesis that cholecystitis glandularis proliferans (Rokitansky-Aschoff sinuses) constitutes a distinct entity and not a variant of chronic cholecystitis.

The 3 characteristic roentgenologic features are: (1) Filling defects at the fundus, sometimes presenting a central pit; (2) a stricture in the body or in the region of the fundus; (3) spaces filled with contrast medium around the periphery of a considerable portion of the gallbladder.

There are no distintive or characteristic clinical symptoms. Refinements and improvements in modern technique make the roentgenologic diagnosis on routine cholecystography possible, though the diagnosis is still infrequently made.—*William H. Shehadi, M.D.*

Skeletal System

Golthamer, Charles R. Duplication of the clavicle; ("os subclaviculare"). *Radiology*, April, 1957, *68*, 576–578. (Address: 14435 Hamlin St., Van Nuys, Calif.)

The author reports a case of a supernumerary bone seen below a normal clavicle on routine postero-anterior chest roentgenograms. This bone, which appeared to be attached to the coracoid process, resembled the normal clavicle. Special views showed the location of this bone to be within the anterior soft tissues about one finger's width below the clavicle. During fluoroscopic examination the medial end of the bone could be moved while the lateral end could not be moved from its relationship with the coracoid process.

A discussion of the origin and development of this anomaly is presented.—*Donald N. Dysart, M.D.*

Smyth, Ivor P. Bilateral osteochondritis of the middle cuneiform bone; case report. *Radiology*, April, 1957, *68*, 575. (Address: Department of Radiology, University Hospitals, Minneapolis 14, Minn.)

The author describes, in a case report, what he believes is the appearance of an osteochondritis found in the middle cuneiform of each foot of a 5 year-old boy who had injured the left foot about six months previously. The boy had an increasing limp favoring the left foot. Roentgenograms showed the right middle cuneiform to be irregular in outline, quite dense and smaller than normal. An abnormal trabecular pattern and fragmentation were also present. The left middle cuneiform showed similar findings but the margins were more regular and the fragmentation less pronounced. An old fracture of the base of the left first metatarsal was also present. Only 1 other similar case was found by the author in a review of the literature.—*Donald N. Dysart, M.D.*

Blood and Lymph System

Anacker, H., Devens, K., and Linden, G. Leistungsfähigkeit und Grenzen der perkutanen Splenoportographie. (Effectiveness and limitations of the percutaneous splenoportography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, April, 1957, *86*, 411–419. (From: Röntgenabteilung der Chirurgischen Klinik der Justus-Liebig-Hochschule, Giessen, Germany.)

Splenoportography is a hazardous procedure, mainly because of hemorrhage from unavoidable rents of splenic parenchyma and capsule. When bleeding cannot be controlled, splenectomy may become necessary without delay. At emergency operations as much as 1,500 cc. of blood has been found in the abdominal cavity. Delayed rupture of the spleen has also been observed from three to seven days following splenoportography in approximately 1 per cent of cases. A fatal outcome has been reported in a few instances.

Nevertheless, the information gained from the procedure outweighs the potential hazards, and it is the impression of the authors that serious hemorrhages will become more or less avoidable with further refinement of technique. Splenoportography should be performed with intratracheal anesthesia immediately prior to a laparotomy. Short-term suspension of respiration will minimize tears of the splenic capsule and pulp.

Absolute indications are: (1) extrahepatic block (thrombosis of splenic vein and portacaval shunt) and (2) intrahepatic block due to liver cirrhosis or obstruction of hepatic segments. Relative indications are: retroperitoneal, pancreatic and hepatic neoplasms, also carcinoma of stomach and gallbladder for determination of operability.

In venous thrombosis the affected vein can now be accurately visualized. Circulation time in the involved area is prolonged two to three times. There is always associated collateral circulation and demonstration of gastric and esophageal varices, in most instances. In cirrhosis of the liver there is also prolongation of the circulation time, backflow of opaque material, a scarcity of portal vein branches and a diminished portavertebral angle. In tumors of the liver, there is crowding of vessels with lack of proper filling, also increased compensatory filling of more distant vessels. For smaller tumors of the liver and the pancreas the method is still unreliable, likewise for determining operability of gastric and gallbladder carcinoma.

Nine roentgenographic reproductions illustrate the various splenoportographic findings, including the collateral circulation.—*Ernest Kraft, M.D.*

General

Goin, Lowell S. Science and solitude versus

clinical consultation: The Carman lecture. *Radiology*, March, 1957, *68*, 319–326. (Address: 658 S. Westlake Avenue, Los Angeles 57, Calif.)

We owe the greater part of our fundamental knowledge of the roentgenology of the gastrointestinal tract to Dr. Russell Carman. He was a great radiologist, a great physician, and a great man. He exemplified the ideal combination of clinician and clinical radiologist. Many of the other pioneers in the field of radiology were primarily good clinicians who did physical as well as roentgenologic examinations.

There is a tendency now for the radiologist to withdraw more and more from the field of clinical medicine. Contact with the patient, and in many instances with the attending physician, is often avoided. Even the details of roentgen therapy are all too often delegated to a technical assistant. It is of course impossible for any radiologist to see personally every patient who comes to his department. This is no justification, however, for the growing practice of never seeing any of them. Unless radiologists function as clinicians and consultants are available for discussion of patients with referring physicians, most of the meaning departs from our specialty.

There has been a gradual but constant increase in the number of general practitioners, urologists, orthopedic surgeons, chest physicians and thoracic surgeons who do their own radiology in their own offices. Most of them do it extremely well within the confines of their own specialty, but they lack the broad experience and the coordinating ability of the well trained and experienced radiologist. There are two sound reasons why the specialist must never supplant the radiologist. The first is that not all patients go to specialists; of those who do, not all are able to select the special fields within which their particular ailments may lie. These constitute a group who may profit greatly by the services of a radiologist. The second reason is that the specialist's attention is directed to his own field to the exclusion of other relevant details.

Unless we are prepared to render service superior to that which a man well trained in his own field can reasonably hope to furnish for himself, we will lose our reason for being and our specialty will perish.—*Arno W. Sommer, M.D.*

Christensen, Wm. R., and Warner, Robert S. The A-V lecture kit. *Radiology*, March, 1957, *68*, 415–(417. (Address: W. R. Christensen, Salt Lake General Hospital, 2033 South State St., Salt Lake City 15, Utah.)

The division of Post-graduate Medical Education of the College of Medicine, University of Utah has been actively investigating methods which will make post-graduate education available to all physicians. Among the techniques which have been tried are: (a) post-graduate lecture series; (b) rural seminars; (c) open circuit television clinics; and (d) audio-visual kits. The last of these methods has been very successful and appears to justify more extensive trial.

The first 2 audio-visual kits are concerned with radiological subjects, namely, "The Accuracy and Reliability of Radiological Diagnosis" and "Radiological Examination of the Chest." The audio-visual kit consists of a single compact box containing: (a) long-playing 33⅓ rpm. records of radiological discussion; (b) script of the discussion including slide cues; (c) 35 mm. slides prepared from Kodachrome transparencies; and (d) a table-top viewer for the slides.

The selection of subjects for the first 2 kits was deliberately slanted toward discussions of a general and basic nature. Each subject was covered by a lecture approximately forty-five minutes in duration. A period of about one year has elapsed since the audio-visual kits were first released. In general, the kits have been enthusiastically received by physicians of the Intermountain Area.—*Arno W. Sommer, M.D.*

RADIATION THERAPY

Decker, David G., Hunt, Arthur B., Fricke, Robert E., and Nelson, Gunard A. Carcinoma of the cervical stump. *Am. J. Obst. & Gynec.*, May, 1957, *73*, 974–984. (From: The Section of Obstetrics and Gynecology and the Section of Therapeutic Radiology, Mayo Clinic and Mayo Foundation, Rochester, Minn.)

From January 1, 1940 through December 31, 1949, a total of 1,451 cases of carcinoma of the cervix were seen at the Mayo Clinic. Of these, 98 were cases of carcinoma of the cervical stump, an incidence of 6.7 per cent. This is almost identical with the incidence of 6.4 per cent found in the series from 1915 through 1930.

The treatment utilized for carcinoma of the cervical stump has been radiation therapy for the majority of patients. In only 5 of 98 cases was any form of surgical procedure performed. This consisted of abdominal removal of the cervical stump and in 3 instances radical removal of the pelvic lymph nodes as well. Eighty-one of the 98 patients were treated by means of combined radium and roentgen therapy.

The usual technique of radiation therapy for cancer of the cervix consists of an intensive divided-dose application of radium with coincident roentgen therapy. In the treatment of cancer of the cervical stump certain modifications must be made in this scheme. In the first place additional care must be used in packing off the radium applicators from the bladder and rectum. This is particularly important, since with the removal of the fundus of the uterus the radium source may be closer to loops of the bowel, and also with the scarring subsequent to pelvic operations the normal anatomic relationships are more

likely to be distorted. The unit of radium treatment is the 50 mg. tube filtered with 1 mm. of platinum. Radium treatments are given two or three times weekly. Total dosage is usually in the vicinity of 4,000 mg. hr. and treatment lasts two weeks. This varies somewhat, depending on the bulk and location of the lesion. The roentgen therapy is administered to two anterior and two posterior pelvic ports. At present, daily treatments of 200 r (air) are delivered at 50 to 70 cm. distance to each of two ports for a total dose of 2,000 to 2,400 r per port, employing 250 kv. (peak) radiation with half-value layer of 1.3 mm. of copper. The technique, nearly to the end of this study, consisted of four daily treatments at 200 kv., 50 to 70 cm. distance, half-value layer of 1.0 mm. of copper, 540 r (air) to each of four pelvic fields following radium treatment. After the course of treatment patients are checked every three months during the first year, with careful pelvic examinations, visualization of the cervix and biopsy if indicated. If the course is satisfactory during this interval the patients are seen twice during the next year and yearly thereafter.

Thirty-five of the cases of squamous-cell carcinoma fell in the relatively favorable Stages I and II and 34 in the more advanced Stages III and IV. It is particularly difficult to stage these lesions accurately, because so often the pelvis is markedly altered by the previously present disease and by the surgical treatment attempted for that condition. These lesions were independently staged by a gynecologist and a radiologist, and the staging shown is a combination of these two independent evaluations. Of 84 patients, 70 (90.5 per cent) were considered to be successfully followed. Fifty-one of these survived for five years or more with an over-all survival rate of 67.1 per cent of the traced patients. If all patients not traced are considered dead of the carcinomas, the absolute survival rate is 60.7 per cent.

With rare exceptions, all of these patients would have been cured by the simple expedient of total hysterectomy at the time of the original operation. With the wider use of the cervical smear for cytology and the recognition of preclinical carcinoma of the cervix, the situation will become more unusual. Carcinoma of the cervical stump remains a problem necessitating the highest diagnostic skill of the gynecologist and the most competent therapeutics of the radiologist.—*Eugene J. McDonald, M.D.*

Roswit, Bernard, Reid, Cyprian B., and Malsky, Stanley J., with technical assistance of Louis Maddalone. A cranial immobilization device for use in roentgen therapy. *Radiology*, March, 1957, *68*, 419–421. (Address: B. Roswit, VA Hospital, 130 W. Kingsbridge Rd., Bronx 68, N. Y.)

A cranial immobilization device for use in roentgen therapy is described. Illustrations show the details of construction and the method of application. The unit has been utilized in hundreds of cases over the past two years and has proved to be entirely satisfactory in clinical practice. Adjustable bi-valve steel yokes provide fixation for the brow and for the occiput. Fixation is absolute and reproducible daily throughout the treatment schedule. It can be attached to the cross-bar of rotational equipment now in general use or to conventional treatment chairs for fixed portal techniques.—*Arno W. Sommer, M.D.*

Murley, A. H. G. Results of radiotherapy in osteo-arthritis of the hip. *Lancet*, April 20, 1957, *1*, 818–819. (From: St. Bartholomew's Hospital, London, England.)

Osteoarthritis has been treated by radiation therapy for relief of pain for over fifty years. There has been no definite agreement on its mode of action and value. Some believe that the radiation causes a hyperemia and improved metabolism. Others think that the blood supply is reduced and that the diminished inflammatory response relieves pain by reducing distention of nerve sheaths.

Sixty-two patients were treated using 1,200–1,500 r from a 150 kv. apparatus in 6–10 fractions in one to two weeks. The few complications included malaise and tiredness in 2 patients and persistent amenorrhea in 1 case, who also stated that the pain was increased by treatment.

One quarter of the patients stated they derived subjective benefit from the irradiation and in only 5 cases did the benefit last longer than one year.—*Charles W. Cooley, M.D.*

Murison, C. A., and Hughes, H. A. Physical measurements on a 4-MEV linear accelerator. *Radiology*, March, 1957, *68*, 367–379. (Address: C. A. Murison, Western General Hospital, Edinburgh 4, Scotland.)

In the linear accelerator described in this article, an electro-magnetic wave with an axial component of electric field is directed along a wave-guide, so that electrons injected into the guide may have their energy increased as they move along with the wave. The electrons attain a final energy of 4 mev. after traveling a distance of one meter, at which distance they strike a 2 mm. thick transmission-type gold target. At a distance of 1 meter from the target, a dose of over 100 r per minute, measured in air, is obtained.

A protection survey was made with the machine operating so as to give a dose rate of 100 rads/min. at 1 cm. depth in a water phantom with its surface 1 meter from the target, with a 20×20 cm. field. This survey demonstrated that the dose received by personnel (for any position of the beam) was considerably below permissible limits.

The physical characteristics of the roentgen-ray

beam, such as symmetry, contamination, and average energy are discussed. Dose distributions for normal and oblique fields are shown. Methods for deriving depth dose for oblique fields are described. Measurements and calculations of surface ionization ratio are compared.

In actual treatment, the dose received by the patient is indicated on a meter of the control desk connected to one of the ionization chambers in the roentgen-ray tube head. A method of calibration of this meter in terms of absorbed dose at a depth of 1 cm. on the axis of the beam in a water phantom with its surface at distances of 80 to 100 cm. from the target for all field sizes (defined at 1 meter) is described. Details of treatment planning are not included, but in general, it is the authors' opinion that the increased depth dose obtainable with the 4 mev. linear accelerator permits irradiating deep-seated tumors with fewer fields than is possible with 250 kv. therapy, and as a result the preliminary work of planning and setting up is easier.

Since integral dose is a factor of some clinical importance in treatment planning, some approximate calculations are made from measured isodose curves. In comparison of a 250 kv. machine operated at 50 cm. focal skin distance with the 4 mev. unit at 90 cm. focal skin distance the much higher depth dose of the latter compensates for the larger amount of radiation scattered outside the beam in the former, with the result that the integral dose per unit applied dose is the same for both machines at a field size of 10×15 cm. The integral dose at 4 mev., however, is only about 60 per cent of that at 250 kv. for a given tumor dose at a depth of 10 cm.—*A. Ralph Watson, M.D.*

Stoll, Basil A. New drugs for irradiation sickness. *Radiology*, March, 1957, *68*, 380–385. (Address: Peter MacCallum Clinic, 485 Little Lonsdale St., Melbourne, Victoria, Australia.)

In continuance of an earlier study of radiation sickness and its management, a controlled trial of a series of new drugs has been carried out at the Peter MacCallum Clinic, Melbourne, Australia. The drugs under trial were chlorpromazine hydrochloride, cyclizine hydrochloride, and pacatal hydrochloride. Their effect was compared with that of pyridoxin hydrochloride, which has been accepted, up to the present, as the most efficacious for the relief of symptoms of radiation sickness.

During the study, the drug under trial was changed weekly to avoid selection of cases. A lactose tablet was used initially as an inert control, but there was no attempt to make all the drugs up in tablets of similar size, shape, and color. The patient was questioned as to alleviation of symptoms one week after the drug was prescribed. If relief was obtained, the drug was continued for that case until the end of treatment. Otherwise, the drug of the second week was prescribed. A total of 212 drug trials were carried out during a period of eighteen months.

Pacatal hydrochloride was shown to be statistically superior to pyridoxin and to all other drugs tested in the relief of nausea and vomiting. Where these symptoms were due to irradiation of the abdomen, chlorpromazine was possibly equally efficacious, but its administration was found to lead to a feeling of sleepiness in some patients.

The mode of action of drugs used in irradiation sickness and possible mechanisms involved in the production of such sickness are discussed. A plea is made for controlled drug trials in the treatment of this condition, and it is pointed out that it is of greatest importance to evaluate the symptoms individually, especially when antiemetic drugs are being evaluated.—*A. R. Watson, M.D.*

RADIOISOTOPES

Moore, Patrick T., and Myerscough, Philip R. Clearance rates of radiosodium from myometrium. *J. Obst. & Gynaec. Brit. Emp.*, April, 1957, *64*, 207–214. (From: The Biophysics Unit, Department of Natural Philosophy, Department of Obstetrics and Gynaecology, University of Edinburgh and Clinical Laboratories of the Simpson Memorial Maternity Pavilion, Royal Infirmary, Edinburgh, Scotland.)

Observations are made by the authors of the clearance of radiosodium injected into the myometrium in late pregnancy. There is a significant slowing of the clearance rate in normal primagravidae past term. There is also a significant slowing of the clearance rate in pre-eclampsia. In cases of pre-eclampsia which are also past term, there is an even more marked reduction in clearance rate.

In pre-eclampsia, clearance rates among the younger women are slower than among older women. This is thought to be due to the inclusion of benign latent hypertensives in the latter group. The reverse is found in normal pregnancy. Among parous women the above differences are most clearly demonstrated, though the numbers of cases in the subgroups are small.

No correlation was found between clearance rates and the degree of hypertension or of albuminuria in pre-eclampsia, nor with placental weight or infants' birth weight in any group of cases.

The range of myometrial clearance rates observed, even in normal subjects, is quite large. This certainly limits the diagnostic value of the procedure, though results are reasonably reproducible in a single patient on separate occasions. But it is during a single test that changes in clearance rate most accurately reflect changes in blood flow.—*Eugene J. McDonald, M.D.*

Montagna, William, and Hill, C. Ruth. The localization of S^{35} in the skin of the rat. *Anat. Rec.*, Feb., 1957, *127*, 163–171. (From: Department of Biology, Brown University, Providence, Rhode Island and Department of Histology, London Hospital Medical College, London, England.)

Skin contains abundant mucopolysaccharides. Chondroitin sulfate is the fraction of mucopolysaccharides which most readily takes up S^{35} and the radioactive particles visualized may represent the site of ester sulfate groups in chondroitin sulfates.

Four female albino rats were injected intraperitoneally with a dose of 15 μc/gm. of body weight of S^{35} labelled Na_2SO_4 dissolved in 0.9 per cent saline containing 0.02 per cent of Na_2SO_4. Eight days prior to the injection, an area on the back of each rat was plucked of its club hairs. Four days later, an area adjacent to it was plucked and twenty-four hours before the injection another area was plucked. The rats were killed respectively at two, four, twenty-four and forty-eight hours after the injection.

It was found that the concentration of S^{35} is maximal in the dermis at four hours and diminishes twenty-four and forty-eight hours later. S^{35} is localized in the ground substance showing greater concentration in the papillary than in the reticular layer. The dermis of the skin with growing hair follicles contains greater amounts of S^{35} than dermis with quiescent follicles. S^{35} is accumulated in coarse granules in the mast cells and the concentration doesn't diminish after twenty-four and forty-eight hours.

Active hair follicles coincident with the appearance of metachromatic substances accumulate large amounts of S^{35} in the dermal papilla and in the outer sheath. Small amounts of S^{35} may be found in the bulb of hair follicles but none are seen in the keratinized parts. The dermal papilla doesn't accumulate S^{35} during the early stages of the hair growth cycle or in quiescent follicles.

The sebaceous glands accumulated very few radioactive particles.—*Charles W. Cooley, M.D.*

Vogel, Howard H., Jr., Clark, John W., and Jordan, Donn L. Comparative mortality following single whole-body exposures of mice to fission neutrons and Co^{60} gamma rays. *Radiology*, March, 1957, *68*, 386–398. (Address: H. H. Vogel, Jr., Argonne National Laboratory, Box 299, Lemont, Ill.)

Experiments during the past several years have been conducted to test the radiobiological additivity of fast neutrons and Co^{60} gamma rays in their acute lethal action on mice. Since high-energy gamma rays and fission neutrons are often encountered together, the question of whether they are additive in producing certain biological effects has arisen. The authors present in this paper the survival data for the 30-day acute period following exposure of a group of 2,414 mice to each of the 2 radiations alone.

It was found that a mortality peak was evident on the thirteenth or fourteenth day following a dose of 929 ± 5 r of Co^{60} gamma rays equivalent to 209 ± 1.1 roentgen equivalent physical of fission neutrons. After neutron doses of 175 to 269 rep, the mortality peak was observed on the fifth or sixth day after exposure. The different patterns of death in these mice indicated that different patterns of injury were involved. The earlier deaths in the postirradiation period (usually associated with severe diarrhea) seem to be due chiefly to an intestinal syndrome while the later deaths, in the acute period, may be largely attributable to damage to the hematopoietic system.

A description of the material and methods used by the authors in these studies is presented and a discussion of their findings, compared to other similar investigations, is given.—*Donald N. Dysart, M.D.*

MISCELLANEOUS

Ehling, U., and Krokowski, E. Schäden durch Röntgenstrahlen am Kaninchenauge in Abhängigkeit von Dosis und Strahlenqualität. (Radiation damage of the rabbit's eye in relation to dosage and quality of roentgen rays.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, April, 1957, *86*, 469–477. (Address: Dr. E. Krokowski, Spandauer Damm 130, Berlin-Charlottenburg, Germany.)

Data regarding the so-called "cataract dose" have varied widely in the literature. Therefore, systematic animal experiments, with varying quality and quantity of roentgen rays, were conducted at the Max Planck Institute to clarify the situation. Altogether, 59 young rabbits were used for the production of radiation cataracts.

The cataracts were divided into the following three degrees: (1) posterior suture clouding, (2) cortical clouding and (3) total cataract. It was found desirable to determine the dose necessary to produce a first degree cataract rather than a cortical clouding or a total cataract.

Physical factors were 180 kvp., 110 kvp. and 60 kvp. The field was round, its diameter being 2 cm. Doses larger than 1,250 rad caused epilation of the eyelid. The cataract dose was slightly higher than the epilation dose. It was found that the absorbed dose was the deciding factor rather than kilovoltage and surface dose. The latter varied from 300 r to 500 r, depending on the half-value layer of the roentgen rays. With fractionation the lens could tolerate a larger dose but, proportionally, not as much as the skin. A regression of the induced cataract could never be observed, which suggests irreversible damage.

The results of the experiments cannot be imme-

diately applied to the human eye, since lens-thickness is larger but thickness of surrounding tissues is smaller in the rabbit. Nevertheless, the additional knowledge gained from the animal experiments can be applied to the prevention of radiation damage to the human lens. The results suggest that the cataract dose is smaller than the available literature indicates. —*Ernest Kraft, M.D.*

Gustafson, P. F., Marinelli, L. D., and Hathaway, E. A. A case of accidental puncture contaminated with Th^{227}; studies on elimination and residual body activity. *Radiology*, March, 1957, *68*, 358–366. (Address: P. F. Gustafson, Argonne National Laboratory, Box 299, Lemont, Ill.)

A few microcuries of $Th^{227}Cl_4$ gained access to the body of a woman laboratory worker at the Argonne National Laboratory as a result of an accidental puncture with contaminated forceps. The clinical management and radioactivity studies instituted are described in detail. On the fourth day a single intravenous injection of CaEDTA (calcium disodium ethylene-diaminetetra-acetate) was given. This increased the urinary excretion of thorium but did not significantly reduce the body burden. The wound and surrounding skin were surgically excised on the fourth day during the administration of the CaEDTA described above. Studies on the total body activity were carried on for about ninety-five days after the injury, and urinary and fecal excretion data on the Th^{227} and Ra^{223} were obtained at intervals up to two hundred and twenty-one days.

Spectroscopic investigation of the gamma radiation *in vivo* and excretion data definitely demonstrated that: (a) thorium in the form of a chloride is readily removed from the puncture; (b) the Ra^{223} daughter is removed preferentially from the wound; (c) thorium is slowly eliminated in the excreta; and (d) Ra^{223} produced in various parts of the body by the decay of Th^{227} is retained in quantity roughly consistent with the equation $0.3t^{-017}$ where t is the time in days.—*Donald N. Dysart, M.D.*

Henley, Catherine, and Costello, Donald P. The effects of x-irradiation on the fertilized eggs of the annelid, chaetopterus. *Biol. Bull.*, April, 1957, *112*, 184–195. (From: Marine Biological Laboratory, Woods Hole, Mass., and Department of Zoology, University of North Carolina, Chapel Hill, N. C.)

The fertilized eggs of chaetopterus pergamentaceus were used to study the effects of roentgen irradiation on living things because: (1) the source was plentiful, (2) the eggs have moderate amount yolk which makes the examination easier, (3) the normal development of chaetopterus has been thoroughly studied and (4) the timetables of development for given temperatures were available.

The fertilized eggs were irradiated beginning thirty minutes after insemination. The following factors were used: 182 kvp., 0.2 mm. Cu inherent filtration, 25 ma.; two roentgen-ray tubes were employed 48 cm. apart with 512 r/min. for doses up to 2,560 r and 16 cm. apart for doses above 3,570 r. Doses from 255 r to 17,280 r were given. Observations were made of both the living and fixed eggs at various intervals after irradiation and of the living trochophore larvae nineteen to twenty-two hours after irradiation.

It was found that doses over 1,020 r resulted in death of most of the larvae by the trochophore stage and that the majority of eggs irradiated with 1,275 r and above showed at least a slight retardation of the first 3 cleavages. Low doses (255 to 1,020 r) resulted in a slight retardation of cleavage and the production of abnormal trochophore larvae which showed severe ciliary defects, cytoplasmic blebs and feeble movements. Cytologic abnormalities included multipolar spindles, chromosome fragmentation and karyokinesis without cytokinesis.—*Charles W. Cooley, M.D.*

SIMEON T. CANTRIL

Janeway Lecturer, 1957